Systems thinking as a novel framework to study overuse injuries in endurance running.

by
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Declaration

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March 2021

Abstract

The chronicity of running related overuse injuries is problematic. Scholars have begun questioning whether the mainstream, reductionist approach to understand the causes behind overuse injuries and interventions to address them, are effective towards healthier athletes. The main aim of this thesis is to describe, develop and evaluate an alternative model to the more traditional, reductionist framework for the management of complex sport phenomena, with a particular focus on running related overuse injuries in the lifestyle and/or recreational runner. Although the systems thinking methodology is diffusing into sport science, the majority of work is of a qualitative nature. Following a design science research methodology, a systems thinking framework was developed as an artefact that is pragmatic, to instantiate systems thinking through computational modelling in sport. The research question is:

> *How can systems thinking and data-driven computational modelling enhance the management and prevention of overuse injuries?*

The systems thinking methodology, simulation modelling and data mining inform the design science research approach at various phases to complete the artefact. The framework consists of two main parts to incorporate both qualitative and quantitative aspects. The concept of the runner as a complex adaptive system, is introduced and is first described qualitatively through systems thinking tools (mainly the ice-berg model and the causal loop diagram) as the combined biomechanical and physiological micro-systems of the runner and their physical, spatio-temporal environment. The qualitative description is transformed to a practical, hybrid simulation model, consisting of an agent-based modelling component that drives a system dynamics component. The transition from linear, cause-effect thinking to closed-loop, dynamic causal thinking was facilitated through the hybrid simulation model. The data parameterising the simulation model are mined from recreational athletes' running watches. Through the simulation, it was possible to show empirically how fundamental solutions and respecting the delays inherent to the runner as a complex adaptive system delivers an athlete who is both fit and healthy. The systems thinking framework supports the notion that both analytical, reductionist thinking and synthetical thinking is required to better understand complex sport injuries at different levels.

Opsomming

Hardloop-verwante oorgebruik beserings is steeds ′n chroniese probleem in sport. Die hoofstroom, reduksionistiese aanslag tot die ontleding en ingryping van oorgerbuik beserings is onlangs in twyfel getrek deur geleerdes en kenners in die veld. Is hierdie aanslag werklik effektief tot gesonder, fikser atlete? Die hoofdoel van hierdie proefskrif is om ′n alternatiewe raamwerk, waarin komplekse sportsverwante verskynsels bestuur kan word, te beskryf, te ontwikkel, en te evalueer. Die fokus is op die hardloop-verwante oorgebruik beserings in die lewenstyl- of ontspanningsatleet. Die navorsingsvraag is:

> *Hoe kan stelseldenke en rekenaarmodellering bydra tot die bestuur en voorkoming van oorgebruik beserings?*

Die stelseldenke metodiek is wel besig om in die sportswetenskapliteratuur te versprei, maar die oorwegende hoeveelheid werk is van ′n kwalitatiewe aard. Deur die ontwerpswetenskap metodiek te volg is ′n stelseldenkeraamwerk as ′n artefak ontwikkel wat pragmaties is om stelseldenke deur middel van rekenaarmodellering te instansieer. Die metodes van stelseldenke, simulasie modellering, en data-ontginning vloei by die verskeie ontwerpswetenskap metodiek fases in om die artefak te voltooi. Die nuwe raamwerk bestaan uit twee dele sodat beide die kwalitatiewe en kwantitatiewe aspekte betrek word. Die konsep, die hardloper as ′n kompleks, maar aanpasbare stelsel, word bekend gestel deur eers kwalitatief beskryf te word deur stelseldenke metodes (spesifiek die ysberg model en die oorsaakskringloop diagram) as die gekombineerde biomeganiese en fisiologiese mikro-stelsels van die hardloper en hul fisiese, ruimte-tydelike omgewing. Die kwalitatiewe beskrywing word opgevolg met ′n omskakeling na ′n praktiese, saamgestelde simulasie model, wat bestaan uit ′n agent-gebaseerde komponent en ′n stelseldinamika komponent. Die oorgang vanaf lineêre, oorsaak-gevolg denke na geslote kringloop, oorsaaklikheidsdenke word deur die saamgestelde simulasie model gefasiliteer. Die parameters in die simulasie model word gedryf deur die data van atlete se hardlopershorlosies te ontgin en te analiseer. Die simulasie kon bewys dat fundamentele oplossings tot oorgebruik beserings, en om die inherente vertragings in die fisiologie van die hardloper te respekteer, beter langer termyn uitkomstes het as kortstondige oplossings wat op simptome fokus. Die stelseldenkeraamwerk ondersteun die standpunt dat beide analitiese, reduksionistiese denke en sintetiese denke op verskeie stelselsvlakke nodig is om komplekse sportsprobleme te verstaan en op te los.

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Acronyms

ABM agent-based modelling

ALS athlete as a living system

BW body weight

CAS complex adaptive system

CLD causal loop diagram

CO cardiac output

CoM center of mass

CVS cardiovascular system

DSR Design Science Research

DSRM design science research methodology

GPS Global Positioning System

GRF ground reaction force

HR heart rate

HRV heart rate variability

MSS musculoskeletal system

NIBIB National Institute of Biomedical Imaging and Bioengineering

OTS Overtraining Syndrome

PMF probability mass function

RCAS runner as a complex adaptive system

rCLD runner's causal loop diagram

RE running economy

RROI running related overuse injury

RW runner's wearable

SD system dynamics

SHM simple harmonic motion

ST systems thinking

STMM systems thinking and modelling methodology

vGRF vertical ground reaction force

VO vertical oscillation

WoD web of determinants

Symbols

$^{\boldsymbol{d}}/_{\boldsymbol{s}}$-ratio	The relationship between the amount of damage stock and structure (unitless)
$\boldsymbol{f}_j$	The stride frequency (cadence) in steps per second for surface type j
$\boldsymbol{F}_s(t)$	The time variable spring force in the legs in reaction to the ground reaction forces encountered during running
$\boldsymbol{k}_v$	Vertical leg stiffness in kN/m
$\sim \boldsymbol{N(\mu,\sigma)}$	Normally distributed with mean μ and standard deviation σ
$\boldsymbol{T}$	The unidirectional flow of time according to the Second law of Thermodynamics
$\Delta\ t$	Time step (or increment) in Newtonian time
$\boldsymbol{x}_m$	In simple harmonic motion, the amplitude is the maximum distance of travel of the center of mass away from the neutral position

Chapter 1

Introduction

Contents

Sport fulfils the purpose of the human body's design to move freely, yet under control. Control implies an athlete is capable of making decisions to promote the optimum outcome. The optimal outcome varies between athletes, ranging from world records at elite level, to maintaining fitness and health through exercise for the recreational athlete.

Nowadays, the frontiers of human performance are taken further through not only the special capabilities of elite athletes, but by the meticulous planning and investments in team work, equipment and infrastructure modulations; owing to a multi-scale, dynamic, complex process. Exemplars are the recent successful (though not officially a record) sub-2 hour marathon by Kenyan elite athlete, Eliud Kipchoge. Aided by an exceptional team of pace makers, careful selection of time and space to optimise the conditions, and a pair of engineered running shoes, Kipchoge completed the distance in 1:59:40 (Keh, 2019). The mix women's marathon record (a female athlete paced by male athletes) was broken in 2019 by fellow Kenyan, Brigid Kosgei in 2:14:04, one minute 21 seconds faster than the previous record set in 2003 by Paula Radcliffe (World Athletics, 2019). However, sport participation across all levels has unintended side effects, one of which is injuries (Malm et al., 2019). In the sport of running, chronicity of the RROI remain prevalent, despite extensive research into prevention and management thereof (Barton, 2018).

1.1 Research background

Scholars have begun questioning the traditional reductionist view on sporting performance and injuries, citing the approach as not being successful towards healthy athletes. Calls for new perspectives are emerging. These new perspectives are founded in the engineering fields and management sciences, namely ST and complexity (Balaguè et al., 2013; Hulme and Finch,

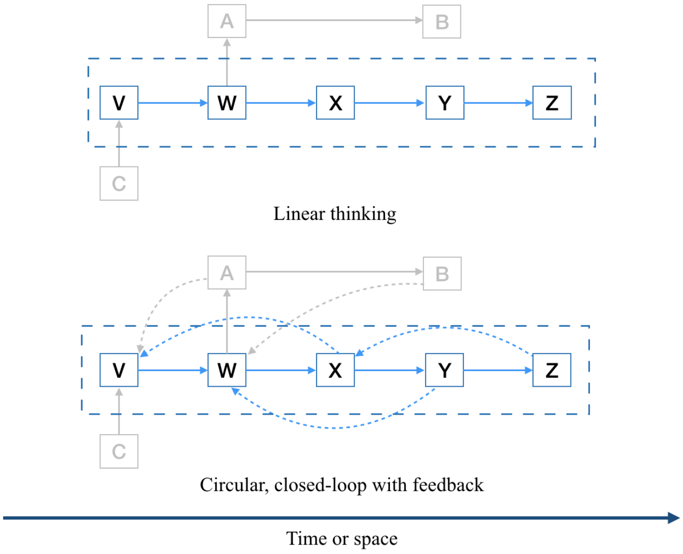


Figure 1.1: Difference between linear and circular, feedback thinking

2015; Bittencourt et al., 2016; Herzog, 2016; Bekker and Clark, 2016). Systems thinking and complexity are diffusing into the literature of sport science, human movement science, public health and other life sciences disciplines. The conceptual, complex sports model from Bittencourt et al. (2016) moves away from finding causes, to finding relations between risk factors (the interacting web of determinants (WoD)) that support emerging injuries. Recently, computational, practical applications of complex ST models of RROI have been demonstrated by Hulme et al. (2018) and Hulme et al. (2019). The data driven computational model is also promoted by Hulme et al. (2019) to complement traditional statistical regression analysis.

Reductionism studies a system (or a structure) by its deconstruction into constituent elements; it is a process of analysis and follows a linear thinking pathway. A system is broken down into hierarchical levels (or subsystems), each with emergent properties of the elements that make up the level (Solomon et al., 2002). The reductionist view analyses complex performance outcomes linked with causal factors in a linear pathway, neglecting varying spatio-temporal scales and isolating risk factors to study its direct effect on outcomes (Bittencourt et al., 2016). However, in living systems, properties found at higher levels are not present in the lower levels (Solomon et al., 2002). Living systems have subtle interconnectedness that becomes sensible only through ST (Senge, 1990). In sports, for instance, a team structure constitute of positions, tactics, set pieces, leadership, collective experience, amongst others, traits not found in the individual athlete who forms part of the team.

Systems thinking interprets complexity and change as dynamic cause and effect over time. Causes and effects are separated through time and space, but in a form of closed-loop thinking, the end (effect) also influences the mean (the cause) (Maani and Cavana, 2007). This is a feedback process, which drives complex system behaviour regardless of the complexity of the individual elements that make up the system (Urze and Abreu, 2014). Influence is circular or multi-directional (see Figure 1.1) (Senge, 1990; Maani and Cavana, 2007), as opposed to linear thinking which is unidirectional. Balaguè et al. (2013) further describe sport not only as a social phenomenon, but also as a real-world deposit of data to analyse and understand human behaviour from a complex ST paradigm. Bittencourt et al. (2016) characterises complex sport systems as open systems with inherent non-linearity, subject to uncertainty and recursive feedback over time. These systems undergo self-organisation to develop patterns of behaviour towards adaptation to the environment and establishment of order. Patterns of behaviour can be classified as either protective or injury risk profiles (Bittencourt et al., 2016).

Herzog (2016) underlines the need for new original ideas and innovative approaches in sport science research, with ST to address complexity, as proposed in Hulme and Finch (2015) and Bittencourt et al. (2016), spearheading the field. Although reductionist thinking have made great inroads to understand cause-effect relationships between variables (Bittencourt et al., 2016), it fails to provide comprehensive explanations for the multi-scale, system wide, complex interactions behind sport injuries (Bittencourt et al., 2016; Hulme and Finch, 2015; Hulme et al., 2019). To understand sport injuries and its management better, there is a need to shift from linear, cause-effect reductionist thinking to causal, closed-loop thinking in sport. However, ST approaches are not meant to substitute traditional, scientific reductionism. Rather, ST may supplement reductionist approaches to advance sports injury research to be system-wide and more comprehensive of all the factors that attribute to injuries (Hulme and Finch, 2015). The ST framework developed in this thesis attempts to address this need; both qualitatively through descriptive ST modelling and quantitatively through data mining and computational tools. Running as a sport is the case study by which the ST framework is set up.

Distance running as a sport is accessible with lower start-up costs when compared to other individual sports such as cycling. It fosters endurance of the body's vital systems and literature is abundant with its favourable effects on general health (Barton, 2018). In the last couple of years, various mobile and wearable tracking technologies that focus on physiological and physical activity metrics to monitor the performance during running in the real world *"has shifted the paradigm of performance enhancements into the hands – or as a matter of speaking – onto the wrists of the athlete, be it on the elite level or the recreational and the lifestyle athlete"* (Vermeulen, 2018, p.1). For the purpose of this study, the wearable technologies used in running will be referred to as the runner's wearable (RW) to include smart watches, activity trackers and running watches.

These self-quantified athletes now present researchers with the opportunity to expand the investigation of the runner's performance in *their* (real) environment, outside of limited and unrealistic laboratory-like conditions or controlled experimental settings (Napier et al., 2017; Passfield and Hopker, 2016). The athlete interacts with the environment, other runners on the road and via the feedback from their RW, even with themselves when they make decisions towards their own fitness goals. They modify their behaviour for optimal performance based on the outcomes from these interactions. Perhaps somewhat unintended, the athlete has promulgated himself as a visible and measurable complex, yet adaptive, system.

1.2 Research opportunity and questions

The main aim of this thesis is to describe, develop and evaluate an alternative model to the more traditional, reductionist framework for the management of overuse injuries. The main research question is:

> How can systems thinking and data-driven computational modelling enhance the management and prevention of overuse injuries?

with sub-questions:

1. *What components constitute a systems thinking framework in sport?*
   Answering this question adds to the theoretical foundation for ST in sport.

2. *How can the systems thinking perspectives be instantiated through computational applications?*
   This question addresses the lack of pragmatism of systems thinking and computation modelling in sport science through its applications on the sport of running.

3. *How can the data from runner's wearables contribute to computational applications in sport?*
   This answer shows how the utility of data from RWs may be extended in sport science.

The outcome of the thesis is expected to grow complex ST and computational modelling both within and *alongside* sport science. Middle to long distance running is the sporting vehicle of choice to deliver this outcome. Recently, the term *gradual onset* injuries have been used instead of *overuse*, however in order to build on the momentum regarding ST and running-related injuries in Hulme et al. (2017b) and Hulme et al. (2018), the thesis remains with *overuse*.

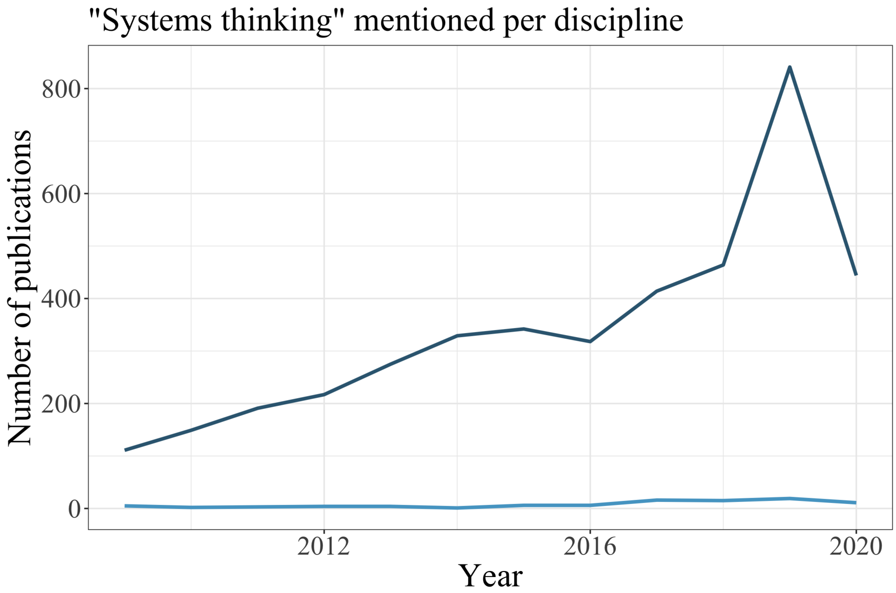
1.3 Research gap

The research study is to fill the gap that is presenting itself at the intersection of three scientific domains: ST, computational modelling and sport science. A first pass literature scan was done on the databases of *Google Scholar* and *Scopus*. The results over the last decade from publications listed in *Google Scholar* are shown in Figures 1.2a, 1.2b and 1.3. Figures 1.2a and 1.2b present the assimilation of literature that included "systems thinking" or "complex systems" in the work, but not necessarily in the title. It is evident that work in healthcare is more abundant and has grown significantly more than sporting publications. Figure 1.3 shows the volume work focussed on complex systems and computational methods (specifically simulations from agent-based modelling (ABM) and system dynamics (SD)) per discipline). The search terms "complex systems", "system dynamics" or "agent based modelling" had to appear in the title, with the sources specific from either health or sport journals. The terms "system dynamics" and "agent based modelling" were pooled together as "computational methods".

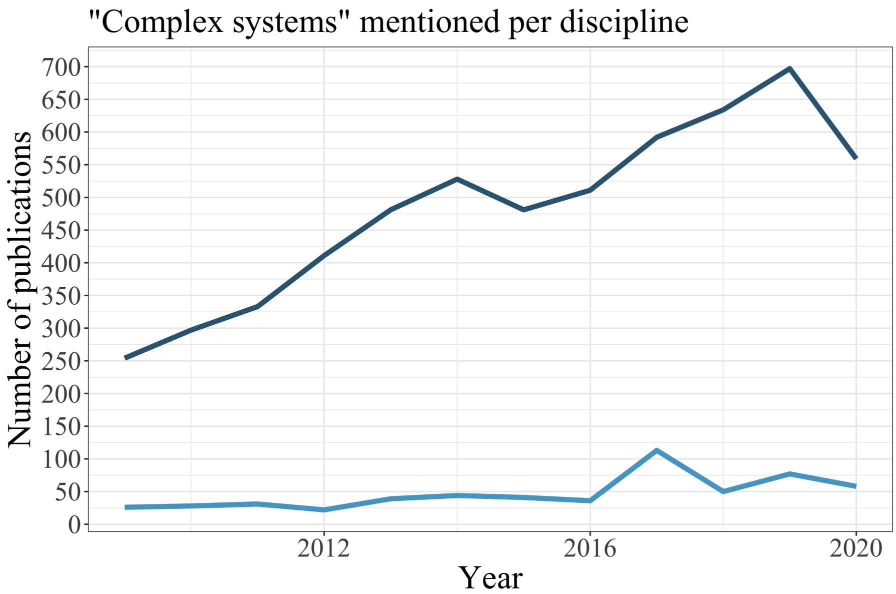
The unfilled space of literature between health and sport sources is evident in Figure 1.3. The paucity in sport literature is noteworthy, with the same search criteria delivering 1520 "complex systems", 1830 "system dynamics" and 244 "agent based modelling" publications from engineering specific sources alone. There is an even spread of complex systems and computational focus in health sources, but it seems that sports literature is somewhat slow to move towards the mathematical and simulation application of complex systems. This observation is in agreement with statements made by authors promoting ST and the use of computational tools in sport (Hulme et al., 2019, 2018), pointing out that work done so far in the field is qualitative and descriptive. The two articles by these authors have demonstrated the practical utility of simulation methods in sport-related injury research. Hulme et al. (2019) presents an SD model in a proof-of-concept format in which running injury development is modelled on a macro system level; Hulme et al. (2018) presents the first agent-based model to relate training load adherence and injury incidence in a synthetic population of runners. The quantified opportunity to extend the knowledge base for ST and computational modelling into sport specific phenomena has presented itself.

Similar searches over the past decade in *Scopus* delivered 165 studies where "complex systems" were mentioned in the research work, although the work included contributions from an array of healthcare professionals. Five studies were found for "systems thinking" in sporting orientated journals from 2007 to 2019. Only one study was found for "system dynamics" in sport for 2009 to 2019, and two for "agent based modelling". The same searches, however, revealed over 6609 articles for "system dynamics", 825 for "agent based modelling" and 539 for "systems thinking" in the general engineering fields alone.

The literature-scoping map proves that ST, computational modelling and sport science have unique and attributable roles to play in the management of complex sporting phenomena. However, at this early stage, the worth of ST and computational tools, specifically simulation modelling, in sport science is nebulous due to the absence of pragmatism. With scholars expressing doubt on the efficacy of traditional, linear causal thinking to prevent and manage complex (and costly) sport injuries, it is worth the effort to take a different approach. Perhaps there is some ‘method in the madness’ of the (novel) synergy between reductionist and abductive thinking in sport. The end goal for the study is a sensible foundation and instantiation for this unique combination of opposites.



(a) Systems thinking



(b) Complex systems

Figure 1.2: Growth of systems thinking and complex systems in work published in health and sporting journals

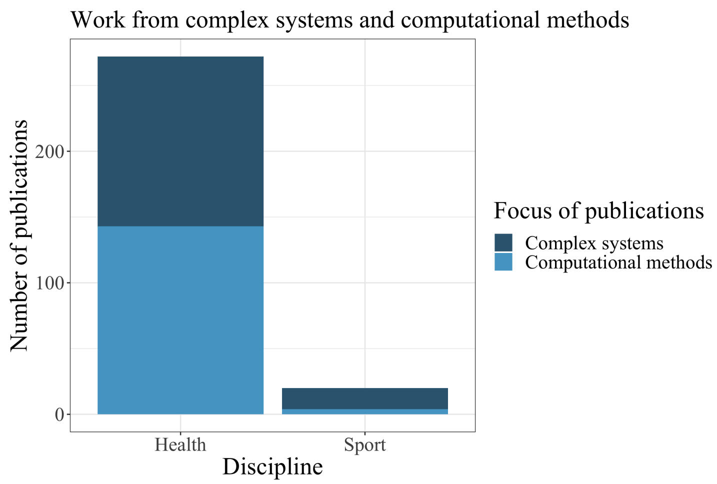


Figure 1.3: In the last decade: work in publication for complex systems and computational methods from discipline specific sources

1.4 Research design overview

The specific objectives of the study are:

1. Contribute to a theoretical and practical foundation for a ST perspective in sport.

2. Introduce and develop the concept, the runner as a complex adaptive system (RCAS), as the combined biomechanical and physiological micro-systems of the runner and their physical, spatio-temporal environment.

3. Instantiate a ST framework in sport science with a practical application in the form of a computational tool to model the RCAS, using ABM and SD.

4. Leverage large data sets generated by RWs (smart watches, activity trackers and running watches) to parameterise the synthetic population in the models.

Figure 1.4 shows the overview of the project's research design and approach. The deliverable (artefact) is a (generic) instantiation for a complex ST framework and its conceptual, computational application in a hybrid agent-based and SD simulation model to study the RCAS with the focus on the emergent incidences of RROIs. The development of the ST framework is divided into three major parts: Part 1 involves theory development, Part 2 contains the ST and data modelling and Part 3 is concerned with demonstration and evaluation of the artefact. For the purpose of this project, the RCAS refers to the lifestyle and/or recreational runner. This deliverable follows on the work by Hulme et al. (2018) to provide a practical departure into complex ST in sport and RROIs.

Furthermore, the simulation model leveraged large data sets generated by RWs to parameterise the synthetic population. This is a unique attribute of the computational application and an advancement for the case of tracking data to be used in sports and, by extension, pervasive healthcare research.

The main purpose of the ST and computational framework is to generate learning in structural leverage in the athlete as a living system (ALS), and not necessarily to predict an injury from a big data set consisting of data points from a large sample size of athletes. Therefore, the idea of the quantified-self as a $n = 1$ sample size from Sands et al. (2017) and Swan (2013) is used, that is the overall, population level sample size consists of a couple of archetype runners, but the dataset per individual athlete is large. With their consent, the data from the acrchetype runners that were used in Vermeulen (2018) was grown in both longitude (larger volume, some dating back to January 2017) and latitude (more variables, by including HR and weather data).

The design science research methodology (DSRM) from Peffers et al. (2008) is the overreaching umbrella in the methodology to create the ST artefact for this study. In creating the ST framework, other relevant methodologies and methods are used to build the models. The six steps in the DSRM are as follows:

1. Identify the problem and motivate: the research questions were arrived at in § 1.2, the gap in the research field is illustrated in § 1.3 and § 1.5 further motivates the creation of the artefacts.

2. Define objectives of the solution: the main objective of the artefact is to provide a framework to support a practical departure for complex ST in sport and the grow the utility of data from RWs.

3. Design and develop the artefact.

4. Demonstrate the artefact in a suitable context.

5. Evaluate the artefact for efficacy and efficiency.

6. Communicate the results to the research field.

Steps 1 and 2 were completed in the introduction. The output from this thesis is mainly focussed on steps 3 to 6. Steps 3 to 5 are guided by the systems thinking and modelling methodology (STMM) and data mining principles. Step 6 is completed as the submission of this thesis, as well as publications prior to submission (see § 1.7).

1.5 Unique contribution

The unique contribution of the research study is an instantiation of ST perspectives in the form of a hybrid simulation model, driven by real-world data. The objectives of the project contribute to research as follows:

1. *Contribute to a theoretical and practical foundation for a ST perspective in sport.* The potential of ST and non-linear computational (and thereby engineering) is expanded into sport science (which perhaps may be a proxy for medical sciences) in collaboration with healthcare and sport disciplines.

2. *Introduce and develop the concept, the RCAS, as the combined biomechanical and physiological micro-systems of the runner and their physical, spatio-temporal environment.* This objective facilitates understanding of sport injuries and performance as a result of behavioural patterns from the interactions between multiple internal and external risk factors, with the eventual outcome of learning how to break the pattern that lead to injury. Running is not the only sport discipline that benefits from the ST model. This thinking paradigm may benefit other sporting codes that are aimed at individual participation and are open to all skill levels, such as cycling, horse riding, swimming, and canoeing to name but a few.

3. *Instantiate a ST framework in sport science with a practical application in the form of a computational tool for the RCAS, using ABM and SD.* The simulation model facilitates the shift in thinking, from isolated, linear cause-effect to a closed-loop, causal structure.

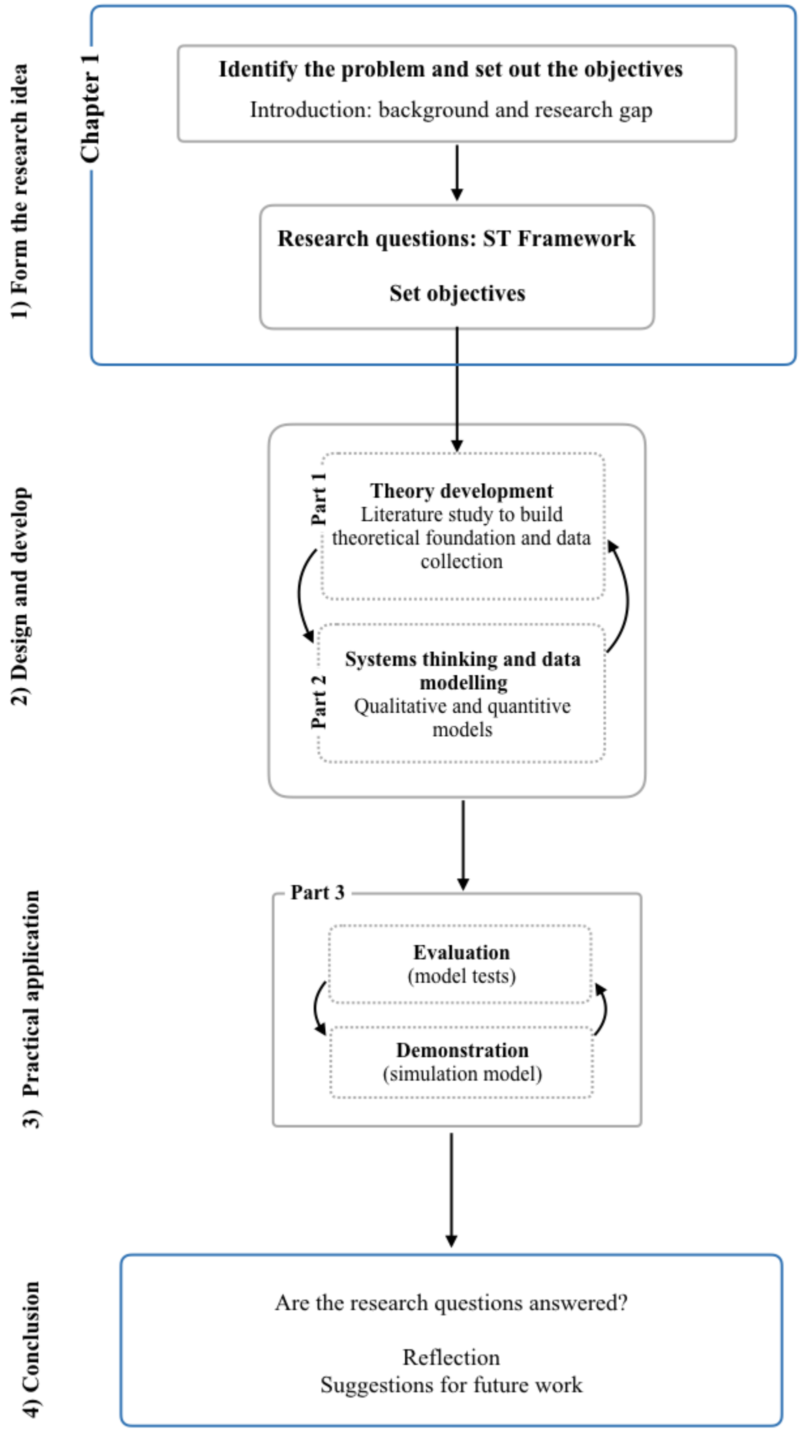


Figure 1.4: Overview of the research approach

4. *Leverage large data sets generated by RWs (smart watches, activity trackers and running watches) to parameterise the synthetic population in the models.* Generous amounts of structured data are currently collected by RWs and stored in the cloud. Researchers have the opportunity tap into this cloud of possibilities to extract meaning and insight that the data can deliver (if treated correctly), creating value that is beyond mere fitness and performance tracking. The utility of the RW data is returned to the athlete as more than descriptive statistics, but as a synthesis of their conditioning over time. The RWs and/or other activity trackers are encouraged to undergo stricter validation to be used as medical devices (this may perhaps generate a new revenue stream for manufacturers). By extension, the case for pervasive healthcare using wearables and mobile health is supported, with physical activity promoted as a self-help method towards healthy individuals. Understanding lifestyle and recreational athletes' behaviour and the path to injury may assist healthcare practitioners in exercise prescription for patients who are capable of independent mobility and opted for running as a fitness intervention during rehabilitation.

This study contributes to the (young) body of knowledge of ST in sport with a practical application thereof to improve the quality management of complex sport phenomena. Sport science can utilise the nomothetic data and findings from traditional reductionist studies as well as the large data sets generated by RWs in applying ST and computational methods to explore and experiment in a safe, virtual environment.

1.6 Layout of the thesis

The introductory chapter presented the research background and the research questions. **Chapter 2 *Research design*** unpacks the research design for the project by describing the methodological processed followed to create the artefacts and data collection procedures. The development of ST framework artefact is divided into three parts, namely: Part I – theory development, Part II – model development, Part III – model demonstration and evaluation.

Part I: Theory development

Chapter 3 *The Athlete as a Living System* describes the biomechanical, physiological and training principles of the athlete in their environment.
Chapter 4 *When the locomotion synergy goes wrong: risk factors and injuries* outlines the adverse effects of too much physical strain, specifically in running. An overview of typical RROIs is provided.
Chapter 5 *The laws of physics applied to running* outlines two important concepts on which the ST modelling and the simulation process is founded upon. The runner is delineated as a spring-mass system in order to quantify the training load from data from the RWs. The Second Law of Thermodynamics is applied to the ALS, specifically the ambiguity of time and the concept of entropy.
Chapter 6 *Methodologies: systems thinking and data-driven computational modelling* unpacks the mechanisms and methods behind ST, SD, ABM, and data mining.
Chapter 7 *Systems thinking and data mining in sport* relates recent advances in sport science research, ST, computational modelling and data mining.

Part II: Model development

Chapter 8 *Data analyses: the digital footprint from the runner's wearable and the weather pattern* provides the analyses of the data mined from the RW, in anticipation

as input to the simulation model.
Chapter 9 ***The generic qualitative and dynamic modelling of the athlete as a living system*** practically applies the concepts of ST to sport and supplies a generic SD model of the athlete's training cycle.
Chapter 10 ***Modelling the runner as a complex, yet adaptive, system*** extends Chapter 9 and instantiates the RCAS, with a detailed, hybrid agent-based and SD model, partially parameterised with input data from a RW, analysed in Chapter 8.

Part III: Model demonstration and evaluation

Chapter 11 ***Evaluation of the simulation of the runner as a complex adaptive system*** evaluates the simulation model with standardised tests to build confidence in the model.
Chapter 12 ***Results from the runner as a complex adaptive system*** provides the various outcomes after experimentation with the simulation model in Chapter 10.
The thesis is concluded in **Chapter** 13 ***Conclusion: synergy is the way forward*** with final remarks and suggestions for future work.

1.7 Publications

Two articles were published from the thesis prior to its submission for examination. The publications are:

- Vermeulen, E., Grobbelaar, S.S. (2019) The potential role for systems thinking in sport. In proceedings for the *7th Annual System Dynamics Conference*, System Dynamics Society South African Chapter, Stellenbosch.
  (This article features the demonstrative, generic SD model in § 9.3. This article received the prize for the System Dynamics Society South African Chapter best student paper 2019.)

- Vermeulen, E., Grobbelaar, S.S., Botha, A. (2020). Conceptualising a systems thinking perspective in sport studies. *Theoretical Issues in Ergonomics Science*, doi: `https://doi.org/10.1080/1463922X.2020.1788662` [Online], published 30 July 2020.
  (This article features the qualitative, descriptive models using ST tools in § 10.1 and § 9.2.)

1.8 Conclusion

This chapter introduced the reader to the research opportunity and provided an overview of the rationale. The research gap illustrated in § 1.3 motivates not only qualitative ST work in sport, but accentuates the lack of practical applications of the methodology in the sports domain. The research objectives in § 1.5 contribute to both theoretical and practical knowledge domains for sport science and ST.

In this thesis, the athlete is analysed into its most important subsystems and elements; then synthesised as a uniquely functioning living system, interacting with time and space in a hybrid SD and agent-based simulation model. This synthetical process provides needed pragmatism in the shift from linear thinking to closed-loop ST. The publications from the thesis prior to examination further prove the value of the research contribution. The comprehensive research design is described in the following Chapter 2.

Chapter 2

Research design

Contents

The DSRM from Peffers et al. (2008) forms the overarching methodological process that guided the project, with the STMM as a sub-methodology to build the qualitative and quantitative models in the ST framework. The data needed for the ST framework were sourced from RWs, a weather data site, and sports domain literature.

2.1 Design science research

Design Science is the how-to knowledge as constructs, methods, models, techniques and/or theory to create artefacts to solve problems or satisfy a set of functional requirements. However, Design Science Research (DSR) is research that creates the missing knowledge for unsolved problems or searches for more effective ways to solve current problems by iterating through a process of design, build, evaluate and reflection (Vaishnavi et al., 2019). In the DSR paradigm, knowledge and understanding of a problem domain and the solution is gained through building and applying artefacts (Hevner et al., 2004). DSR may be defined as the process by which knowledge is used to design and create an artefact that is useful. This artefact is then rigorously analysed for its effectiveness during evaluation; whether it works and why it does or does not work. Understanding is gained during the evaluation phase. This understanding is returned to the knowledge base of the discipline. The value of the research therefore does not lie in the design, but in the process of the design. Design in itself is not a knowledge generating process; however, the meticulous evaluation and analysis of the effectiveness of a designed artefact in its intended environment is a knowledge generating process and may be considered research (Manson, 2006).

2.1.1 Outputs from design science research

> *"This community would welcome effective artefacts that enable such problems to be addressed? Constructs by which to think about them, models by which to represent and explore them, methods by which to analyse or optimize them, and instantiations that demonstrate how to affect them."*
>
> (Hevner et al., 2004, p.85)

The outputs from DSR (Table 2.1) are created artefacts, in the form of constructs, models, methods, frameworks, instantiations, architectures, design principles, and design theories (Vaishnavi et al., 2019). A DSR artefact may conceptually be any designed object with embedded research contributions, setting it apart from other routine designed artefacts (Peffers et al., 2008).

Table 2.1: Outputs from design science research (adapted from Vaishnavi et al. (2019))

Output	**Description**
Constructs	A domain's conceptual vocabulary.
Models	The set of statements or propositions expressing the relationships that exist between the constructs. Models are problem and solution statements: suggestions for how things are or ought to be.
Frameworks	Guides (either real or conceptual) meant to provide support or guidance.
Architectures	The high-level structures of systems.
Design principles	Central principles and concepts to guide design.
Methods	How-to knowledge: the sets of steps used to carry out tasks, such as an algorithm. Methods are plans to manipulate the constructs to realise the solution statement (model).
Instantiations	Situated implementations, where, in certain environments, the abstract artefacts such as constructs, models, and methods are operationalised. Instantiations may precede completed constructs, models or methods, for the basic reason that understanding would not be generated without a working artefact.
Design theories	A set of statements prescribing how to execute tasks to achieve an objective. Other abstract artefacts are usually included in such a theory.

The output from this research project is an instantiation of a novel approach to sports overuse injuries. The ST framework consists of models and methods, operationalised in a simulation model.

2.1.2 The knowledge contribution

Gregor and Hevner (2013) describe two distinct knowledge bases to which DSR may contribute through the creation and evaluation of artefacts: the descriptive base, i.e. the 'what' knowledge is about phenomena, natural laws and regularities; the prescriptive base refers to the 'how-to' knowledge. Descriptive knowledge is leveraged to justify theories related to the research problem, while the existing artefacts in the prescriptive base that have solved similar research problems are investigated. A DSR project draws from both these bases to form a baseline of existing knowledge and the novel contribution of the new artefact (see Figure 2.1). As part of the DSR process, knowledge is returned to either base during analyses (or evaluation) of the artefact (Manson, 2006). The use of the artefact may result in new regularities or previously unwitnessed phenomena when applied in its environment (Hevner et al., 2004), while the artefact contributes to the how-to knowledge base for why it works (or does not work) (Manson, 2006).

In Figure 2.2, Gregor and Hevner (2013) outlined the knowledge contribution matrix for DSR artefacts. The axes deal with the maturity of the problem or application domain and the solution domain. *Inventions* are artefacts addressing new problems with new solutions. In this quadrant, it is also possible that a problem description does not yet exist, frustrating the validity of a new artefact to solve it. *Improvements* are artefacts that address a well-known problem but with a new solution. This new artefact must prove advantageous above the current artefacts, in terms of efficacy, efficiency or some other objective. *Exaptation* adapts a well-known solution to a new problem. In these three quadrants, DSR makes knowledge contributions. *Routine design* uses existing, well-known solutions to solve well-known problems, and therefore no new research knowledge is contributed. In this research project, contributions are made to the prescriptive knowledge base. The ST framework is an improvement on and a supplement to the current strategies to solve RROIs.

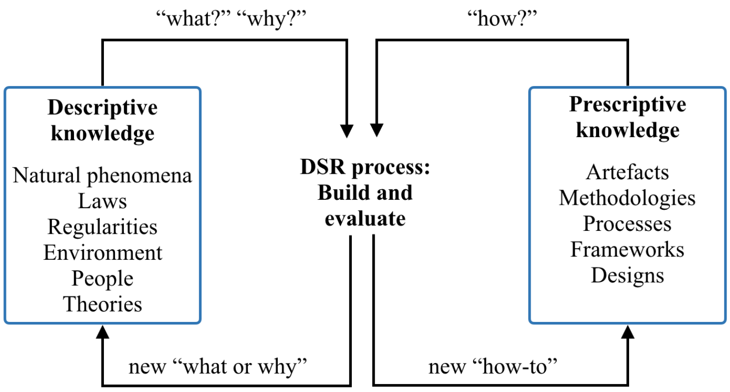


Figure 2.1: Knowledge bases and flow of contribution

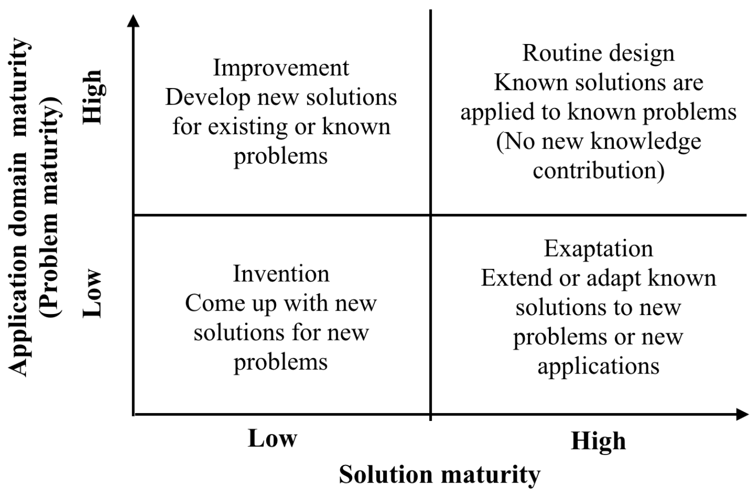


Figure 2.2: Design science research knowledge contribution matrix, adapted from Gregor and Hevner (2013).

2.1.3 A process and cognitive model for design science research

The general process for a DSR project consists of problem description, building or creating an artefact, evaluation thereof and communication if warranted. Several processes have been suggested in the literature to create frameworks that may standardise the approach. The DSR process from Vaishnavi et al. (2019) is shown in Figure 2.3. The process starts with *awareness of the problem*, followed by the *suggestion* phase in which abductive thinking promotes the searches in the knowledge bases to understand the problem and find a solution for it. However, if none is found or those that do exist are inadequate, a new or improved artefact is created in the *development* phase. The artefact is then evaluated according to specifications during *evaluation*. Deductive findings from the evaluation feed back to *suggestion* and *development* iteratively to inform further suggestions, changes to the design and subsequent new developments. This *circumscription* process allows knowledge to flow to the knowledge bases.

Conclusion follows development when the researchers decide that the effort has been sufficiently evaluated and the DSR project comes to an end. The write-up of the results and what was learnt (on what worked and what did not work) to solve the problem may be communicated to the research field as *design science*. Reflection and abstraction of general themes underlie the cognitive processes undertaken to communicate the artefact or the outcome of the DSR project. The newly created (or improved) artefact is added to the prescriptive knowledge base as 'how-to' knowledge, while new observations or understanding of phenomena when using the artefact may be added to the descriptive knowledge base (Vaishnavi et al., 2019).

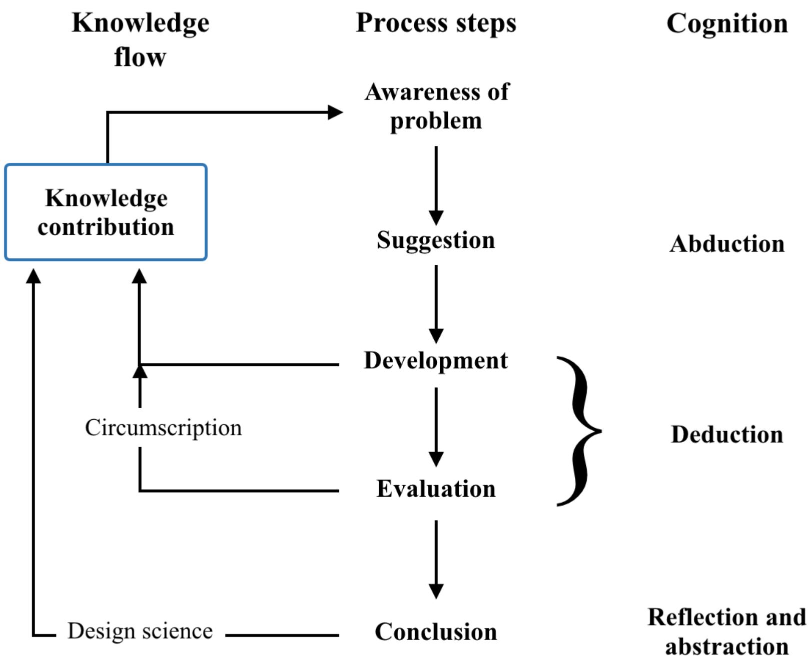


Figure 2.3: Design science research process, adapted from Vaishnavi et al. (2019).

2.1.4 Artefact evaluation guidelines

To evaluate the utility of artefacts and contribution to the knowledge base, guidance is taken from key questions articulated in Hevner et al. (2004):

1. How does the new artefact's utility compare to existing artefacts? If existing artefacts are adequate, then new artefacts are irrelevant.

2. Does the artefact map adequately to the real world? This question addresses relevance and methodological rigor.

3. Is the artefact capable to solve the problem? If not, it does not have utility.

4. Has the artefact been demonstrated for its utility? Demonstration must provide the basis to its claims of utility and contribution to the knowledge base.

5. What are the implications for research, in terms of the problem, the artefact and its utility? Clear implications warrant publication or communication.

The simulation model of the ST framework has been evaluated and reflected upon against these criteria, and is tested and evaluated using well-known and established tests from Sterman (2000) and Forrester and Senge (1980).

2.1.5 The cycles of a design science research project

Figure 2.4 shows the extended four-cycle model from the known three-cycle model of the DSR approach. The fourth cycle, *change and impact*, integrates the artefact with the dynamic, broader environment in which the artefact is utilised (Drechsler and Hevner, 2016). The *relevance* cycle relates the need for the artefact in the real world, or within its immediate application context through requirements and field-testing. There are problems that need solving or opportunities to improve artefacts. An artefact is field-tested to determine its capability to solve the problem. Feedback from the field evaluation aids in the iterative design of the artefact. The *design* cycle consist of rapid build-and-evaluate activities, in order to refine the artefact before deploying it for field-testing. The *rigour* cycle draws from the knowledge bases to ground the methods in the design and application of the artefact. Knowledge is returned to the knowledge bases through evaluation and testing of the artefact (Hevner et al., 2004).

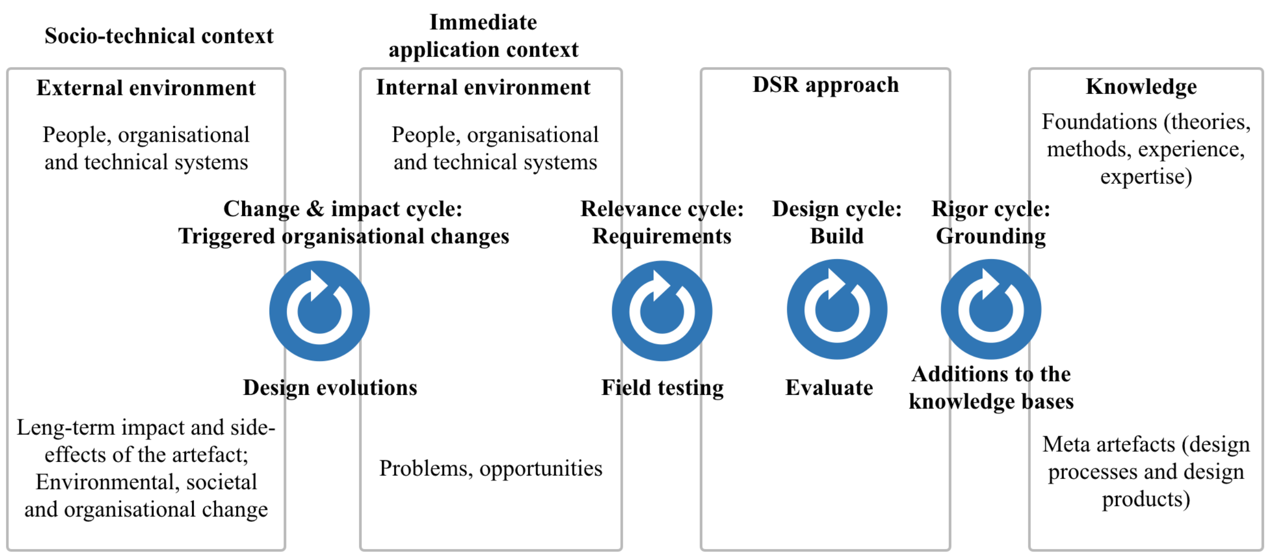


Figure 2.4: The four-cycle design science research model, adapted from Drechsler and Hevner (2016).

2.2 The design science research methodology

Peffers et al. (2008) developed their DSRM to improve adoption of the DSR approach in information science research; however, the approach is as applicable to the research project presented

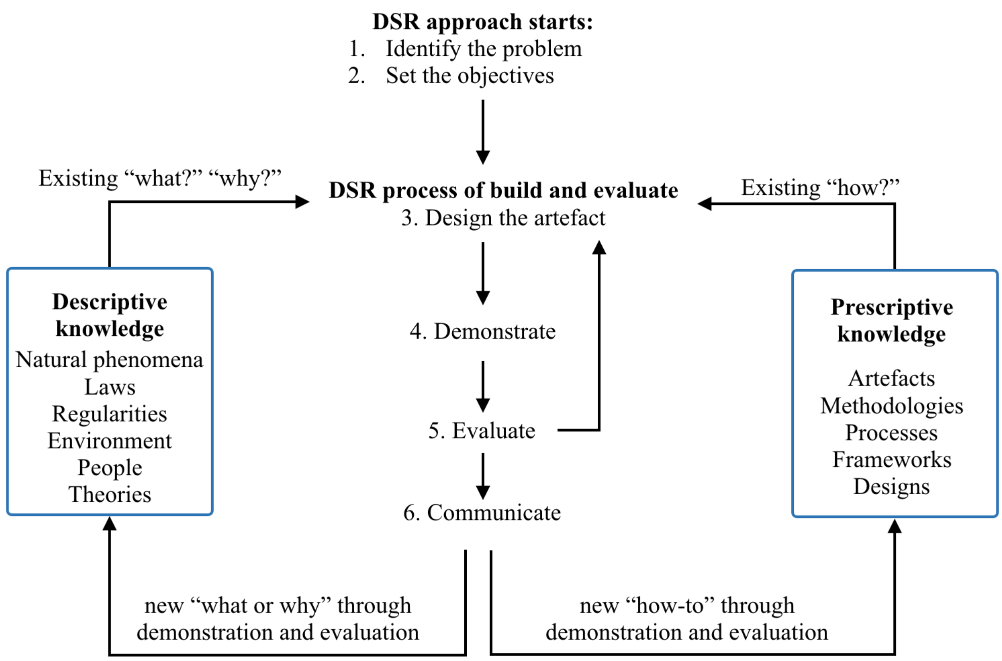


Figure 2.5: The design science research methodology and the flow of knowledge

in this thesis. The six-step DSRM is an adaptation of the process steps provided in Vaishnavi et al. (2019) and is the overreaching umbrella in the methodology to create artefacts for this research project. The main breakaways from the process in Figure 2.3 involve *awareness of the problem*, *suggestion* and *development*. In the DSRM, the first step is deconstructed into two steps: *identification of the problem* and *defining objectives of the solution*. *Suggest* and *develop* from Figure 2.3 are joined together as *design and development*. The six steps are as follows:

1. Identify the problem and motivate.
2. Define objectives of the solution.
3. Design and develop the artefact.
4. Demonstrate the artefact in a suitable context.
5. Evaluate the artefact for efficacy and efficiency.
6. Communicate the results to the research field.

Steps 3 to 5 are iterative: evaluation of the artefact generates new knowledge that may re-inform the design of the artefact, necessitating another demonstration and evaluation. The iteration may extend back to step 2, if it requires a redefinition of the objectives. However, this cycle must not continue indefinitely, and at some point, the results from evaluation are considered as 'good enough', prompting write-up of the artefact and findings in the last step, communication (Manson, 2006). The process is depicted in Figure 2.5 with the flow of knowledge.

Identify the problem

The research problem must be defined and a solution justified. The problem definition done well assists the researcher to atomise the problem and develop an effective artefact. Knowledge of both the descriptive base of the application domain and the prescriptive base is required to describe the scope of the problem and justify a suitable solution, i.e. an artefact that will solve the problem (Peffers et al., 2008).

Define objectives of the solution

The objectives of the solution are deduced from the problem definition and from the knowledge of what is feasible and possible. Objectives may be metric or non-metric (Peffers et al., 2008).

Design and develop the artefact

The artefact's functionality and architecture are determined and the actual artefact is created, drawing from the descriptive and prescriptive knowledge bases. The artefacts are constructs, models, methods, instantiations, design theories or novel properties of technological, social or informational resources (Peffers et al., 2008).

Demonstrate the artefact in a suitable context

The artefact is practically demonstrated to solve instances of the problem. This step must provide proof that the artefact works and is capable to provide the utility required to solve the problem. Demonstration may be transdisciplinary and consist of experimentation, quantitative, qualitative, in a case study, in a simulation, ethnography or in some other form of appropriate practical application (Peffers et al., 2008).

Evaluate the artefact for efficacy and efficiency

Assess the artefact's capability to solve the problem by comparing the results from the demonstration to the initial set of objectives. Appropriate metrics and rigorous analyses techniques are used to measure the artefact's efficacy and/or efficiency. Evaluation may include any suited empirical evidence or logical proof of the artefact's performance. Outcomes from this step may necessitate an iteration back to step 3 to inform the design to improve some aspect of the artefact, or continue to write-up the results and communicate the new knowledge (Peffers et al., 2008).

Communicate the results to the research field

The research community, industry and other users of the artefacts must be informed of the problem, its relevance and the newly created artefact. Communication to the application and research field includes the artefact, its utility, the rigour of the design, efficacy and efficiency (Peffers et al., 2008).

2.3 The systems thinking and modelling methodology[1]

The ST methodology is concerned with the externalisation of mental models and consists of two main phases: qualitative modelling and quantification of the system. The systems thinker's language of communication is mostly visual, with qualitative modelling such as networks, the causal loop diagram (CLD), structural diagrams, system archetypes (Goodman, 1991), the ice-berg model, rich pictures and cognitive maps (Maani and Cavana, 2007), whereas quantification consists of time-series (behaviour of time of system variables), relational graphs, network

[1] part of this section was published in *Theoretical Issues in Ergonomics Science*, July 2020

analysis and computer simulation (micro-worlds of the real system) (Maani and Cavana, 2007; Kirkwood, 2013).

The STMM steps are set out by Maani and Cavana (2007), and consist of problem structuring, conceptual qualitative modelling (primarily the CLD), quantitative dynamic (primarily simulation) modelling and scenario planning. With problem structuring having been taken care of in step 1 of the DSRM, steps 2 and 3 of STMM inform the DSRM steps 3 and 4 as a sub-methodology to create and build the ST instantiation (see Figure 2.6). The qualitative modelling steps constructed the ice-berg model for the runner (including the WoD as used in the complex sport model from Bittencourt et al. (2016)), the generic CLD, the runner's causal loop diagram (rCLD), and mapped systemic problems in sport injuries to system archetypes. The dynamic, quantified model builds on the qualitative CLDs in a hybrid SD and agent-based simulation model, using as input the data mined from the RW, the weather website *MeteoBlue*, secondary data from literature, and illustrative, hypothetical data.

2.4 Data mining

Data mining can be defined as the process of collecting, cleaning, processing, and analysing data in order to extract knowledge from it (Aggarwal, 2015). In this research project, data were mined from three athletes' RWs and a website containing granular weather data of cities (*Meteoblue*, `https://www.meteoblue.com`).

The systematic extraction, processing and aggregation of data from RWs is as follows:

1. Download the Global Positioning System (GPS) container files, (file extension *.tcx*) from the athletes' online fitness profiles (on *GarminConnect*). The GPS container files include the GPS location (latitude and longitude), the altitude, a date-time stamp, distances covered, speed, cadence, and heart rate for nearly each second over the duration of the run activity. Other data directly extractable from the application are: age, sex, height, VO_2 - max (if provided by the device). The athlete could also choose to provide this data on the consent form.

2. The athletes were interviewed on their injury history (main events) and asked to report any injuries during the course of the study.

3. Extract the data from the container files in *R* (using the *XML* library), clean, and write to the *SQLite* database.

4. Extract the data from the database and perform necessary clean-ups, calculations, and aggregations using own code or built-in algorithms.

5. Visualise data and extract patterns for input into the simulation model.

The quantitative models include the behaviour over time analyses and cluster analyses of data mined from the RWs.

To quantify external, environmental parameters in the simulation model, weather related data is available from the *MeteoBlue* website for granular, hourly historic data. In excess of 1 075 968 data points were extracted from the *MeteoBlue* weather site over the course of one year (June 2019 to end of May 2020) for selected South African cities and towns where the participating athletes run (Cape Town, Pretoria, Johannesburg, Bloemfontein, Durban and Belfast). The data contain the following weather variables for every hour of the included year: temperature, relative humidity, mean sea level pressure, total precipitation (high resolution), total precipitation (low resolution), snowfall amount (high resolution), snowfall amount (low resolution), total cloud cover, high cloud cover, medium cloud cover, low cloud cover, sunshine duration, shortwave radiation, wind direction, wind speed, and wind gust. Data is downloaded as either *.csv* or *.xlsx* files, processed in *R* and written to the *SQLite* database.

2.5 Research design

The detailed research design for the project is shown in Figure 2.6. The various parts of the research process are laid out in more detail than earlier in Figure 1.4. The chapters where the work feature are indicated on the figure. There are mainly four phases of the research project, guided by the elements of the DSRM from Peffers et al. (2008):

1. Forming the research idea (identifying the problem and objectives and setting the research questions).
2. Design and development of the artefacts, through theory development and model development.
3. Practical application of the artefacts (demonstration and evaluation).
4. Conclusion, where the artefact is reflected upon.

The main research question necessitated three sub-questions, sub-RQ 1 to sub-RQ 3. Part 1 answers sub-RQ 1 by critically evaluating the theoretical foundations of a ST framework. Answering sub-RQ 2 and sub-RQ 3 is concerned with creating the ST framework, which is done in Parts 2 and 3 (see Figure 2.6). Parts 1 and 2 are completed in an iterative manner. Part 1 is concerned with theory development from the sports domain, ST, computational methods (simulation modelling) and data mining, i.e. the descriptive and prescriptive knowledge bases are explored to understand the problem, existing solutions and analyse the suggested solution. The components of the ST framework are identified. Part 2 develops the models for the ST framework, both qualitative and quantitative. These models are built from the concepts and literature explored in Part 1. The STMM from Maani and Cavana (2007) informed the process taken in Part 2 to create the qualitative and quantitative models. Data mining specifically underwrites the process of cluster analyses and behaviour over time. Part 3 demonstrates and evaluates the ST framework through model testing and experiments, also an iterative process. Simulation model testing was done guided by the model tests from Sterman (2000). The simulation model of the ST framework is evaluated against the DSR criteria from (Hevner et al., 2004) (outlined earlier in § 2.1.4 *Artefact evaluation guidelines*). Concluding the research project is a reflection on the ST framework, how the components relate and suggestions made for the way forward.

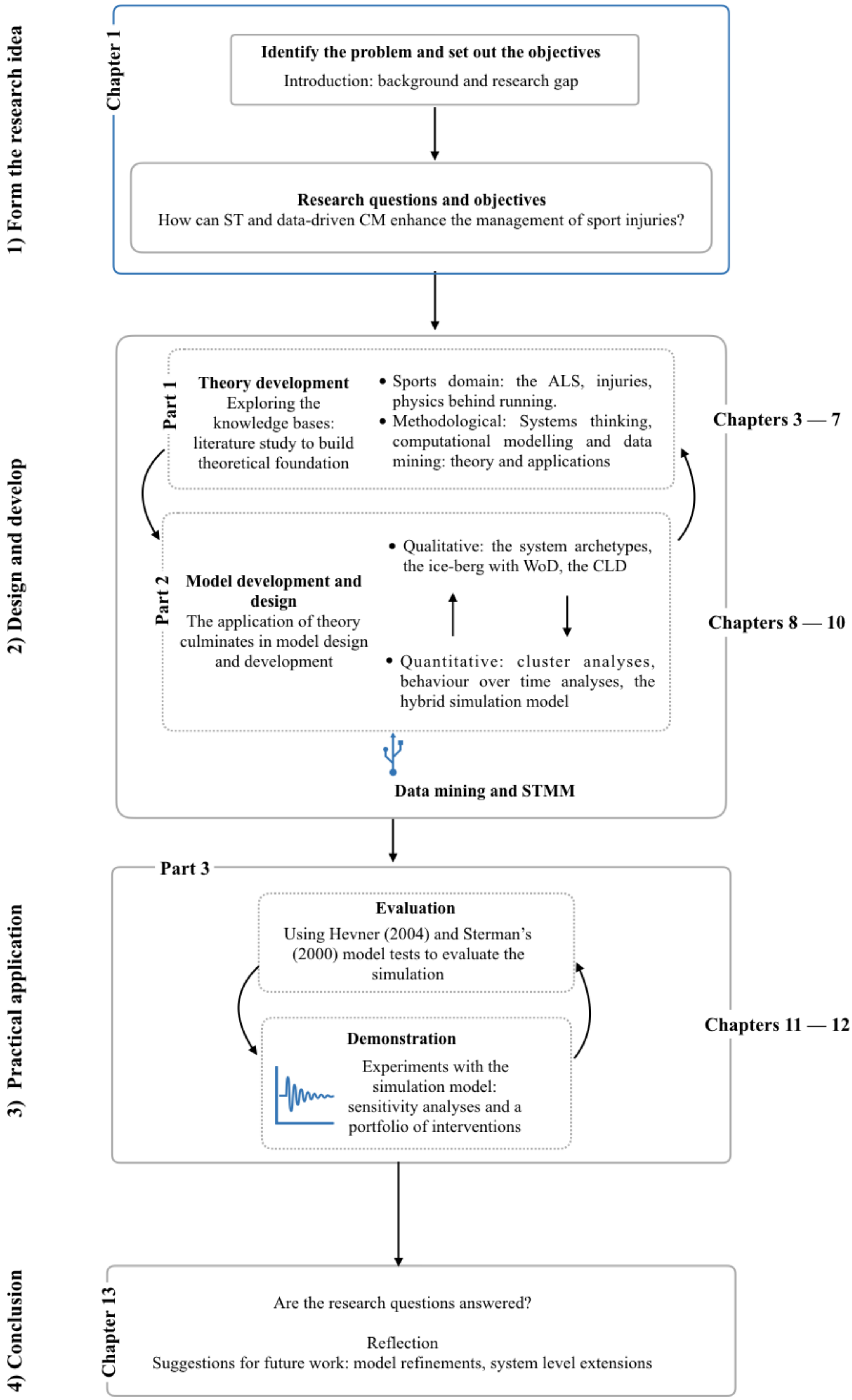


Figure 2.6: The research design for the project

2.6 Research methodology

The mixed-method research execution consists of the following tasks and sub-tasks:

1. A comprehensive evaluation of the literature on ST, computational modelling, and sport science to capture the state of the research fields.
   (a) Develop an advanced theoretical and practical knowledge base of ST and computational modelling, particularly in sport.
   (b) Analyse the characteristics of a complex adaptive system (CAS) in conjunction with applicable computational methods. The computation methods, namely ABM and SD, are analysed theoretically alongside practical applications (in sport and healthcare) in order to develop a sound mathematical background of the methods. Other computation methods, such as machine learning, are explored in the literature as alternative and/or supportive solutions to the problem.
   (c) From (a) and (b), synthesize the current research state for ST, computational modelling and sport science.
2. Data acquisition and organisation thereof into a database. The reader is referred to Appendix B for a detailed methodology on data collection from RWs. The study received ethical clearance from the Stellenbosch University Research Ethics Committee: Social, Behavioural and Education Research (SBER), with project number 10124.
   (a) Download and extract the running data from the athletes' online fitness profiles. The GPS container files include the GPS location (latitude and longitude), the altitude, a date-time stamp, distances covered, speed, cadence, and HR for nearly each second over the duration of the run activity.
   (b) The athletes were interviewed on their injury history (main events), and ask to report any injuries during the course of the study. This was an open-ended question as part of the consent form. The injury did not have to be diagnosed by a doctor.
   (c) Extract the relevant environmental (more specifically, weather) data from a publicly available dataset, *MeteoBlue*.
   (d) Organise the runners and environmental data into a database for analysis, aggregation and eventual input for model operations.
3. Analysis of the relevant data (behaviour over time) and transformation into useful mathematical representations as input into the computational model. The time line of the data collection is January 2017 (or the earliest available data from the athlete) up to September 2019. An exploratory analysis of the data from the RW included the following:
   (a) A clean-up procedure using an outlier detection algorithm.
   (b) Analysis of the distributions of the variables.
   (c) ANOVAs
   (d) Generating behaviour over time and interaction graphs.
   (e) Discretisation and classification using cluster analyses.
4. Synthesis of the ice-berg framework of the RCAS.

(a) Develop an in-depth understanding of the biomechanics, physiology and pathological pathways during running as a physical activity and RROIs as well as the external spatio-temporal factors that impact running output, such as road surface, gradients, altitude, and the weather.

(b) Out of (a), identify the internal and external elements in the WoD for the RCAS.

(c) From (b), construct the network of the WoD and establish the strength of the links.

(d) Lay out a pathway from the network of WoD to running behaviour (the physical output) and the event of a RROI.

(e) Abstract the WoD and their pathways to behaviour into the most important determinants and present it in an ice-berg model.

5. Transformation of the RCAS into qualitative causal loop and structural diagrams.

   (a) Map the system archetypes from the ST literature to general sport behaviour.

   (b) Map a high-level training cycle as a CLD, then extend this training cycle to the rCLD by combining the system archetypes. The variables in the rCLD are extracted from the WoD.

   (c) Identify the goal-seeking points and the various feedback loops in the WoD: polarity, re-enforcement, balancing and dominance structure.

   (d) Identify the tuning parameters of the system. These parameters serve as levers in the computational models in order in introduce variation in the self-organisation of the WoD.

   (e) Present the rCLD and the structural diagram as a comprehensive, visualised system.

6. Construct the hybrid agent-based and SD model from the CLD to quantify the self-organisation processes between internal and spatio-temporal entities and analyse the regularities (risk or protective profiles). The two main outcomes from the simulation are improved fitness or an injury.

   (a) Decide on outcome granularity: which events or behaviours from the CLD must be analysed on agent level, and which on population (or systems) level?

   (b) Use the elements, feedback loops and goal-seeking points from the CLD to identify the agents and their attributes in the model.

   (c) Identify the states in which the agents will function and the transitions between the states.

   (d) Generate the population of athletes with their attributes and decision rules. Parameterise the attributes and lever variables with the extracted data sets.

   (e) Link agent behavioural patterns (regularities) in the agent-based model to aggregated outcomes (stocks and flows) in the SD model. That is, aggregate the agent behaviour into system level behaviour.

7. Evaluate the hybrid model in terms of sensible and counter-intuitive outcomes, realistic computational time, user practicality and parsimony.

8. Experiment with the hybrid model by executing the simulation under varying conditions with the adjustments of levers. Observe behavioural changes and identify the most influential patterns that lead to injuries or improved fitness.

Tasks 2 to 8 were conducted in tandem as an iterative process to develop the framework and the computational application, with some overlapping of the literature review process. Data collection and analyses (tasks 2 and 3) precede the qualitative modelling, because data are required to set up behaviour over time and graphical frameworks for the RCAS and the hybrid modelling application.

The RCAS is presented in the subsequent chapters, created from the methodological principles and tools presented here: the ice-berg framework decomposes the runner into its constituent elements; system archetypes are used to describe patterns of behaviour seen in sport; the runner's path to injury is depicted in a CLD as the growth-and-underinvestment archetype. The CLD of the RCAS is transformed into a hybrid simulation model to perform experiments and define optimal leverage for long-term change. Furthermore, unsupervised machine learning algorithms assisted the modeller to prepare the data from RWs as input for the simulation model. Although machine learning may also be used to study patterns of behaviour, it lacks the mechanical transparency of ABM and SD, which is needed to gain understanding in the interrelated behaviour of the RCAS.

2.7 Conclusion

This chapter unpacked the research approach and the methodologies whereby the artefact were created and evaluated. This research project merges the disciplines of engineering, data mining, and sport science. The DSR approach simplifies and provides rigour to an otherwise multifaceted and complicated, transdisciplinary research project.

Part I

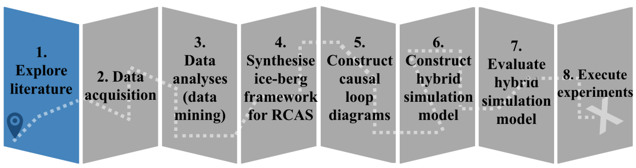
Theory development

Chapter 3

The Athlete as a Living System

Contents

This chapter formulates part of task 1 from the research methodology.



"...by seeing wholes we learn how to foster health."

– Peter Senge, 1990

Neither the beating heart on its own, nor isolated well defined muscle structures, will displace the athlete's body through space – only through the interactions between these elements of the system is the athlete able to move. The runner must first be understood as an athlete in order to later formulate the RCAS. The specific themes from the running-related literature presented here were selected to substantiate the total-body involvement that running as a tri-planar activity demands. The selection is further based on the available data points (or attributes of running) from the RW *.tcx* container files. The major attributes are cadence (link to biomechanics and running gait), distance and pace (training load), and HR (the cardiovascular response). These attributes may be linked with the immediate environment through the geographic location provided in the GPS coordinates (longitude and latitude). The environment presents with different temperature and other weather-related phenomenon, which influence how the runner's body deals with environmental heat stress through thermoregulation. Figure 3.1 relates the available data to the selection of literature.

Running is an endurance sport that requires consistent and stable supply of fuel and oxygen to muscles by means of the cardiovascular system (CVS) over a prolonged period of time, as well as the simultaneous removal of waste products, including heat through thermoregulation.

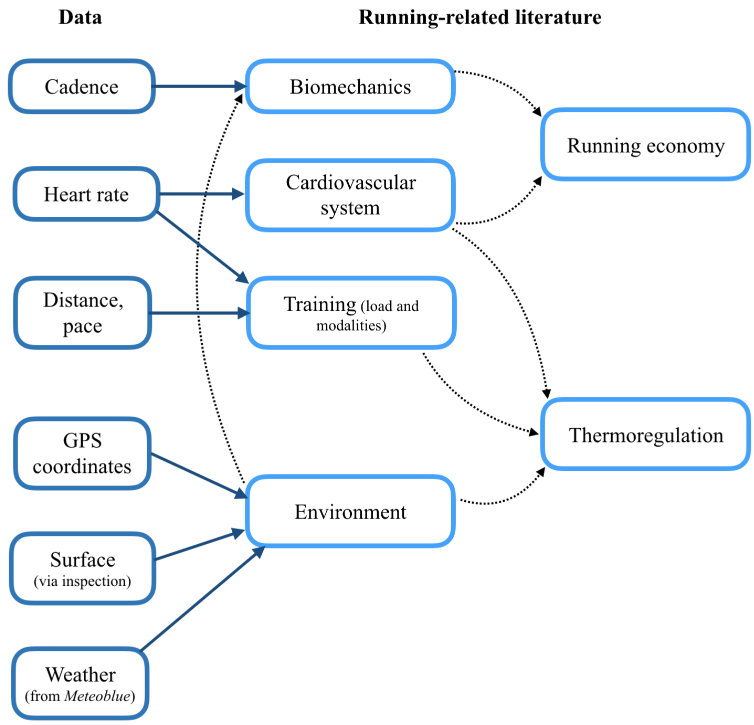


Figure 3.1: Relation between available data and running-related literature.

Biomechanics is required to understand how the body's levers (limbs, joints and muscles) function as a machine in motion to safely transport the body as a single mass over the ground. Running induces physical, dynamic loading of the body's structures at higher velocity than walking, and therefore requires strength and resilience to deal with the cyclical stress and strain. Training methods are applied to induce the required physiological adaptation for resilience to deal with cyclical stress (both in physical loading and the demand for circulation). Finally, running economy (RE) provides a summary for how the body optimises its physiological processes to support a stable CVS and a resilient athlete. Excluded from the literature review are genetics, nutritional aspects, altitude, slopes (road inclination), running equipment, and psychological aspects. Although part of the greater body of literature for long distance running, the anticipated modelling of the RCAS did not include these aspects in order to maintain parsimony and a focus on driving the simulation with tracking data available from the RWs.

3.1 The athlete's body in locomotion

The healthy synergy between cardiovascular fitness, neuromuscular pathways and musculoskeletal structures is what constitute an effective and efficient ALS. For movement to occur, the ALS must function almost like a machine. Levers work in a synchronised pattern to propel the body in a direction. Energy is supplied from physiological processes to drive these patterns. The mutual dependency between biomechanics and physiology is what defines human locomotion. This section serves as a rudimentary grounding of the basic concepts behind human locomotion. Detailed theory may be found in the sources mentioned here.

3.1.1 Biomechanics

Figure 3.2 provides an overview of how biomechanical concepts are subset in order to describe the motion patterns in the complex musculoskeletal system (MSS) fully. Biomechanics constitute *"the principles and concepts that govern the body's response to active and passive stresses on its segments"* (Levangie, 2005, p.4).

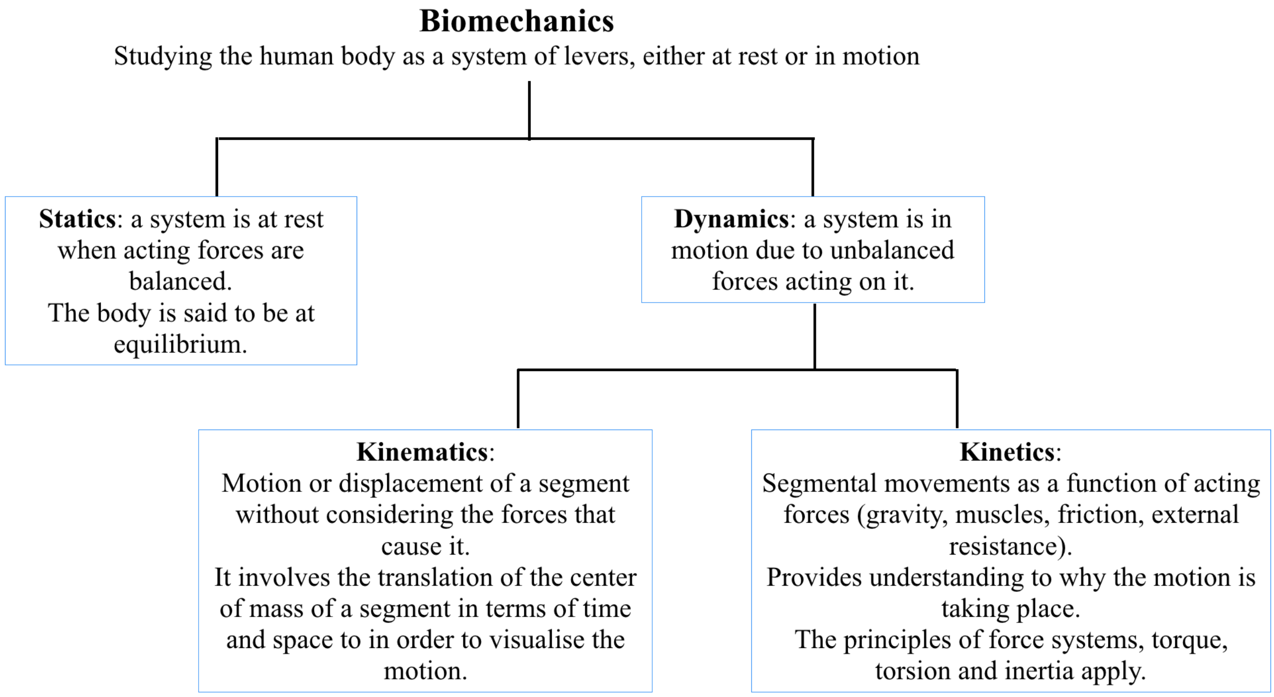


Figure 3.2: Breakdown of the components of biomechanics, adapted from Levangie (2005).

Four main structures are involved in the biomechanics of the MSS: bony segments, ligaments, joints and muscles. A link is formed when two or more bony segments are connected with ligaments at their endpoints. Joints consist of a link and the cartilage tissue covering the bony segments, with a capsule to encase them (Levangie, 2005). Muscles attach to bony segments: a muscle has an insertion on one segment, then cross over a joint and attach to the adjacent segment (Agur and Dalley, 2005). The bony segment and its joints will be collectively referred to as levers, with the attached muscles applying force to generate movement.

Skeletal muscle

Gravity is an omnipresent force on earth, under which the body will not be able to support itself without skeletal muscle action, let alone perform tasks. There is no artificial substitute for human muscle (in contrast with many prosthetic limbs and joints that have been created), making them indispensable in the ALS (Chleboun, 2005). Muscles attach to bones through their associated tendons, together they are referred to as the musculotendinous unit.

Skeletal muscle *initiates and maintains* movement through concentric contraction (shortening of muscle under load) and *control* movement with eccentric contraction (lengthening contracting: a muscle is lengthened under load). Muscles work together in agonistic pairs across joints. Through concentric muscle action on the segment, the agonist muscle generates torque, initiating movement and maintaining it through the range of motion of the joint. The antagonist muscle opposes the torque and slows down the angular velocity through eccentric muscle activity. Muscles are doing positive work during concentric contraction (as the bones move closer together) to lift a load; negative work is done during eccentric contraction (when the bones move away from each other) because work is done *by* the load *on* the muscle (Chleboun, 2005).

The stress-strain curve

The structures in the MSS are subject to the physical stress imposed by the external loads, resulting in strain (deformation) in the segments, joints and muscles. The same principles from the stress-strain diagrams in the studies of the strength of materials found in engineering applications (Hibbeler, 2011) are therefore applicable to the MSS. The amount of deformation in a material will differ due to different material properties. The nominal stress experienced by materials is a function of the load, P and its original cross-section area, A_0 in Equation 3.1:

$$\sigma = \frac{P}{A_0} \text{ (units:} \frac{N}{mm^2} \text{ or Pascal, } Pa) \tag{3.1}$$

Nominal strain is related to the change in length of the material under stress (δ), and its original length, L_0, that is:

$$\epsilon = \frac{\delta}{L_0} \text{ (unitless)} \tag{3.2}$$

The stress-strain curve plots the stress against the strain, seen in Figure 3.3. The curve is subset into a linear (elastic) and non-linear (plastic) portion. In the elastic region, the material's units are resilient to applied stress, such that it is able to return to its original length when the force is removed. Opposite behaviour is found in the plastic region, where the fibres undergo permanent deformation. The failure point is found in the plastic region, which is when the material has been deformed beyond its plastic capacity and subsequently tears or ruptures. Hooke's law describes the relationship between stress and strain in the elastic region (linear) in Equation 3.3, analogue with the formula for a spring with load constant (or the stiffness coefficient) k, on which a force F is applied to compress or stretch it by a distance x:

$$F = kx \text{ (i.e. the spring)} \tag{3.3}$$

From Equations 3.1 and 3.2, the linear portion of the graph in Figure 3.3 is termed the constant of proportionality, referred to as the modulus of elasticity or Young's modulus, E. The modulus of elasticity is essentially the slope of the elastic region, i.e. the rate of change in stress σ for every unit increase in strain, ϵ:

$$\sigma = E\epsilon \tag{3.4}$$

E and k represents the same concept: the resilience of a material to resist deformation and return to its original form.

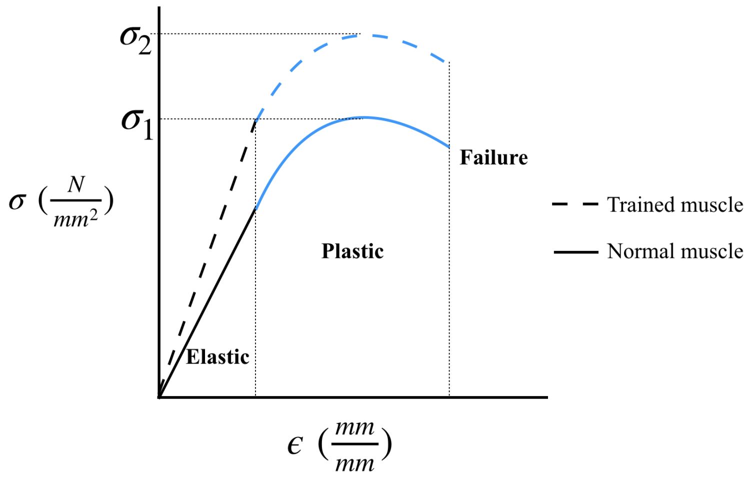


Figure 3.3: The stress-strain curve (or the load-deformation curve)

In biomechanics, the stress-strain curve is referred to as the load-deformation curve, yet the same principles hold (Chleboun, 2005):

1. Deformation differs for muscles, ligaments, joints and bone as they consist of different biological materials and subsequent have other mechanical properties.

2. Fibres return to their original length in the elastic region, but experience progressive deformation in the plastic region, indicating microfailures of tissue fibres. Injury occurs along the plastic region, with full rupture of soft tissue or fracture in bones, at the failure point.

However, the MSS is part of a living system and can alter the mechanical properties of its units, which will change the stress-strain behaviour. These adaptations are the drivers behind training philosophies (Robi et al., 2013).

The motion-stability complex

During sporting activities, the athlete's body may be in either a static state (for instance on the start line) or in in a dynamic state, moving in multiple directions. The athlete must be able to alternate between these phases in quick succession, demanding complex interactions between the body's segments. One such example can be seen on the start line, whilst waiting for the start signal and then propelling forward; another is evident in the stop-start-stop movements such as in team structured ball sports. The athlete must be able to change direction (on the ground, whilst airborne or in water) in an instant and often unexpectedly. These alternations are not as pronounced in distance running as in fast paced team sports, however perturbations in balance caused by changes in pace, surface, slopes and negotiating road obstacles all require the body to adjust its biomechanics in order to both stabilise and generate motion at the same time (Honeycutt and Nichols, 2014; Vernillo et al., 2016).

Joints serve the same purpose as joints found in buildings and machines. Joints allow (and disallow) movement of segments *relative* to each other: in some cases, the joint must allow movement of the section, but should also stabilise and/or support the segment during motion or in a fixed position (Curwin, 2005). Take the movement of the lower limb during running as an example: the knee should flex and extend while the foot is airborne, from toe-off through to the next footfall. During the swinging motion, the two main lower limb segments (the femur-tibia complex) must be controlled to remain within a safe range of motion, such as to prevent dislocation. Upon the next footfall, the same joint must now stabilise the femur-tibia complex relative to each other during the ground contact phase. These active stabilisations, both during swing and weight transfer, are now an interactive function between joint surfaces, ligaments and muscles.

The interactions between muscles, joints, ligaments and bony segments are the drivers behind kinematics. It is therefore important to comprehend and appreciate the biomechanics in identifying intrinsic risk factors, more so their interaction and the patterns of movements produced. Movement in the ankles seems to be far removed from movement in the trunk, especially while seated, but during running, ankle and trunk motions are linked via the kinematic and kinetic chains. The kinematic chain defines how the system of levers transfer motion to both adjacent and satellite joints in a predictable way (Curwin, 2005); and in Brukner and Khan (2006) the kinetic chain is described as the sequencing of levers to actively perform a task. A proximal joint (closer to the trunk) must be stabilised for a distal joint (further from the trunk) for its lever to move through its range effectively (Brukner and Khan, 2006). That is, one (or more) joints serve as a stable base for another joint to perform its intended function.

In the open kinematic chain, joints are not fixed and free to move independently from the other joints in the chain. Non-weight-bearing movements, such as knee flexion while the opposite knee is grounded, consist of open kinematic chains as the ankle and the hip do not necessarily move, but will depend on the range through which the knee is flexed. In closed kinematic chains, a joint at the end of the chain is fixed and movement at the joint naturally causes movements in connected joints. Joints in a weight-bearing position undergo closed kinematic motion; for example bending the knees while the ankle is fixed to the ground automatically translates to motion in both hips and ankles (Curwin, 2005).

Open and closed kinematic chains work in synergy during walking and running to stabilise the body and move it forward in a predictable manner. The hip is stabilised at the pelvis to allow for a safe weight transition over the grounded foot (closed kinematic chain) while the airborne leg is swung through during walking and running (in an open kinematic chain). An excessive 'pelvic drop' is evidence of poor stabilisation at the hip with the grounded knees and ankles now inheriting the responsibility to deal with most of the landing and transition forces (Olney, 2005).

Variables to describe the structure of the musculoskeletal system

The structural attributes of the MSS are described in terms of the shape(s) of joints and bones, related to their attached skeletal muscle and ligament properties. Variables include joint angles (the alignment or relative positioning of bone segments to each other), ligament length and laxity, bone length and density and muscle length and size. Table 3.1 provides some vocabulary defined for this thesis to describe deviating characteristics of the units in the MSS and its impact.

Table 3.1: Variables to describe the MSS

Structural discourse	**Impact on the MSS**
Malalignment of joints	Disruption in the optimal transfer of forces and torque production
Ligament laxity	Joint moves beyond the safe, controlled range; risks dislocation and instability of the lever
Muscle dystrophy and/or atrophy	Weakness to generate force for movement; unable to stabilise joints.

Curwin (2005) states that in the ideal MSS, the form of a unit follows it function. However, both internal (biological) and external drivers of everyday life result in degrading (or improving) attributes of the MSS. The 'form follows function' thereby has a bi-directional undertone: the form (physical attributes) of the MSS impacts the functioning of its levers to carry out active tasks, while the intended function of the levers demands a certain form of its components in order to execute tasks.

3.1.2 The running gait

Gait is described as the translation of the body's center of mass (CoM), by means of *balanced* rotary movements of its segments through space and time (Olney, 2005). An ineffective and inefficient gait stems from unbalanced movements – when kinetics is demanded from deficient kinematics. Deficient kinematics encompasses both decreased and uncontrolled range of motion at the joints, that is, it is a function of restricted movement at the joints or insufficient stabilisation of joints.

The running gait cycle differs from the walking cycle mainly in two different aspects. First, the double-stance phase in walking (when both feet are grounded) is replaced with a flight phase, with both feet being airborne and during no phase of running are the feet both grounded (Adrian and Cooper, 1995). Second, the base of support during running is reduced when compared with walking (Olney, 2005). Figure 3.4 illustrates the running gait cycle, which describes the biomechanics during its two phases: stance and swing. The term 'footfall' constitutes the three touchdown mechanisms in which the feet make contact with ground: heel strike, mid-foot and front-foot landing. Once the leg has lifted the toe from the ground (end of stance), this leg enters the swing phase (*A* and B in Figure 3.4). Just before the next footfall of the same foot, the entire body is airborne for a short while; this is the flight section of the swing phase (*B* in Figure 3.4). The involved leg is transferred through the air to pass underneath the body and come forward, then down towards the ground and enters the stance phase upon footfall. The opposite leg is in stance phase while the swinging leg is airborne and passing underneath the body. The stance phase consists of footfall to toe-off of the same foot (*C* to *E* in Figure 3.4). During stance the body's weight is transferred over the grounded leg.

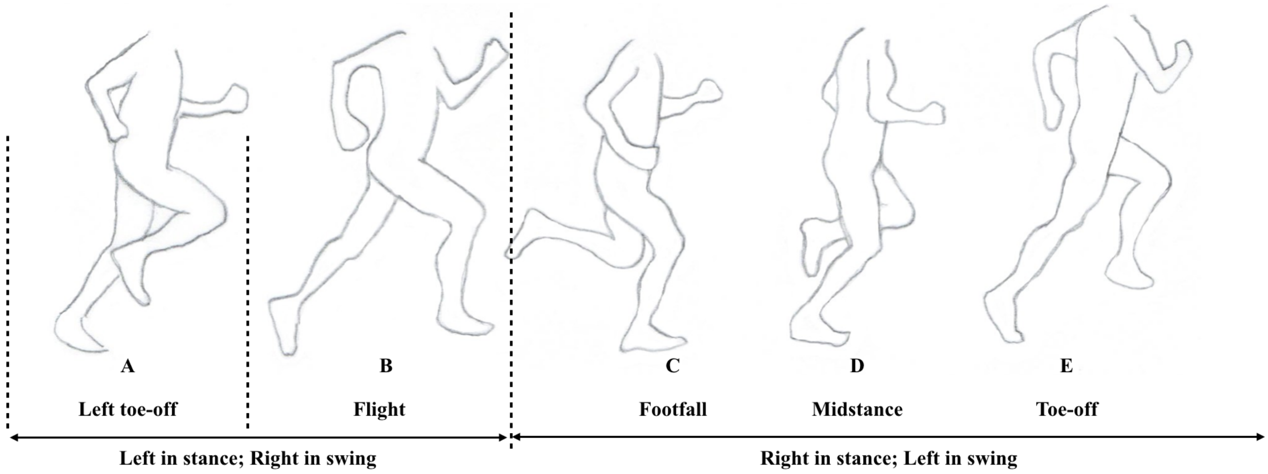


Figure 3.4: The running gait

The action of running demands increased balance, more muscle strength and optimal joint range of motion when compared to walking. More work is demanded from the muscles, with main reasons being (Olney, 2005):

- The CoM must be raised higher during the flight phase (kinetic energy in the MSS must be converted to potential energy) and
- Stabilisation actions are more intense at the joints (both in the air and in stance).

The body has a longer travel from its highest position in the flight phase back to the ground and at higher speed, demanding greater shock handling and energy distribution from the MSS in less time than walking. Shock is generated by the force acting from the ground on the MSS. Upon footfall, the foot applies forces onto the ground and in turn, the ground applies forces to the foot. The force applied to the foot is termed the ground reaction force (GRF) and is of concern in both walking and running gait studies. The GRFs are equal in magnitude but opposite in direction of the forces generated by body weight (BW) acting on the ground (Moore, 2016; Olney, 2005).

During the stance phase, from heel strike to toe-off the GRFs fluctuate as the foot's centre of pressure (i.e. its contact point with the surface) moves along a characteristic pattern (Olney, 2005). GRFs during walking fluctuate between 70% and 80% of BW and reach maximums of 120% BW during the stance phase. However, running may impose GRFs up to 200% BW at the centre of pressure and may reach 250% of BW during the gait cycle (Olney, 2005).

The vertical ground reaction force (vGRF) is the vertical vector of the GRF acting on the body. The vGRF-time waveform patterns are utilised to study the loading rates of the lower limbs by vGRFs during stance (Clark et al., 2017), expressed as $BW.s^{-1}$. The vGRF–time waveform represents the fluctuating vGRF over time during the stance phase. During running, average vGRF loading rates on lower limbs may range between 30 to 300 $BW.s^{-1}$ for injured and non-injured runners, with 70 to 80 $BW.s^{-1}$ considered excessive (Shih et al., 2019). Shih et al. (2019) found an average loading rate of 65.8 ± 17.3 $BW.s^{-1}$ in a cohort of 40 runners where leg stiffness was associated with vGRFs. The vGRF reached 2.5 times BW at 50% of the stance phase in a group of female runners who had a history of tibial stress fracture; mean loading rate (standard deviation) of 78.97 (24.96) $BW.s^{-1}$ were recorded for the stress fracture group compared to 66.31 (19.52) $BW.s^{-1}$ in a control group (Milner et al., 2006).

Some important running gait variables are cadence, stride length (Heiderscheit et al., 2011; Schubart et al., 2014; Adams et al., 2018), vertical oscillation (VO) (Adams et al., 2018; van der Worp et al., 2016; Souza, 2016), ground contact time (Wang et al., 2012), stride angle, foot inclination at ground contact (Santos-Concejero et al., 2014), stride width (cross-over gait) (Brindle et al., 2014; Meardon and Derrick, 2014), and foot movement (supination and pronation) (Brukner and Khan, 2006; Nigg et al., 2015). These variables are the product of the runners unique gait pattern that emerges from the interactions within the MSS. A runner adjusts their biomechanics to arrive at the optimal gait pattern for them, discussed further in § 4.4.2 *Gait retraining: running is a skilled activity*.

Vertical oscillation is the vertical excursion of the CoM between the stance and flight phase and is associated with loading rates of the lower limbs; higher loading rates lead to injuries such as stress fractures (Adams et al., 2018). Closely related in gait patterns, cadence and VO have been shown to have different effects on loading rates. Whereas both an increased cadence and lowered VO reduced loading rates, a lower VO was more effective to reduce the peak vGRF and only a higher cadence reduced the breaking impulse (Adams et al., 2018). Furthermore, Wille et al. (2014) suggested increases in cadence as a strategy to reduce the VO, the loading rate of the GRFs and decrease the braking impulse. The interaction between VO and cadence is an important aspect of running biomechanics, both for locomotion purposes, and to protect against injury (see § 4.4.2 *Gait retraining: running is a skilled activity*). The dynamic modelling (to follow in Chapter 10) focuses on the runner as an oscillating spring-mass system modelled with simple harmonic motion (SHM), whereby VO represents the amplitude of the runner's CoM and cadence is the frequency of the wave-form function.

3.1.3 The cardiovascular system

The CVS is not directly impacted in the dynamic modelling of the ALS or the RCAS, although it is important to understand its supportive role to the MSS to enable and sustain locomotion. The CVS is responsible for the circulation of a very complex liquid: blood. The CVS consists of the heart as a pump, with the arteries and veins forming an intricate network of vessels through which blood is transported throughout the body. Arteries terminate in the capillary bed, a network of small diameter vessels surrounding and infiltrating the tissues. Blood exits the capillary bed through veins, back to the heart. Blood carries oxygen, hormones, protective substances (such as immune cells) and nutrients (fuel substrates) to cellular tissue for life processes to be delivered at capillary level. At capillary level, it takes up from tissues the waste products from cellular metabolic activity (amongst others, urea, lactic acid and CO_2) and transport these to excretory organs. Furthermore, it regulates the body's temperature by means of blood flow adjustments to the skin (Meyer et al., 2002; McArdle et al., 2010).

Circulation and oxygen consumption

Circulation is vitally perpetual; failure in any of its components results in either starvation of much needed substances or dangerous build-up of waste products. Cell damage is inevitable with prolonged disruption in circulation (Meyer et al., 2002). During locomotion, the MSS works in synergy with the CVS to facilitate muscle work and soft tissue adaptations. The CVS sustains all the locomotive activities in the MSS by providing oxygen and fuel for energy production in muscle cells, then removing waste products from the cells and finally regulating the raised internal temperature. Some special attention is given to cardiac output (CO), maximal oxygen consumption, VO_2-max and oxygen extraction, $a - \bar{v}O_2$-diff. CO is the amount of blood that is pumped by the heart in one minute, ml/min and is a function of HR and stroke volume, discussed in more detail later on. Although enough blood may reach the tissues, the oxygen in the blood must still be extracted for it to be useful. The indicator of the amount of oxygen extracted is the difference between arterial O_2 content and mixed-venous O_2 content, presented as $a - \bar{v}O_2$-diff. The VO_2-max is maximum oxygen consumption in one minute per kilogram body weight ($ml \cdot kg^{-1} \cdot min^{-1}$), a measure that reflects an athlete's aerobic capacity (McArdle et al., 2010). Increases in CO, oxygen extraction and oxygen consumption are characteristics of improved cardiovascular fitness, because blood is reaching the tissues and meeting the demand sufficiently, and oxygen that is available is extracted and consumed to match the demands for energy production from the working MSS. A compromised CVS will not be able to support the MSS, leading to soft tissue damage, poor performance and possible injury.

The heart and its autonomic regulation

The heart at the centre of the CVS is regulated through the myocardium input from the autonomic nervous system. The autonomic nervous system's role during and after training (the physical load enough to cause a level of physiological stress), is to maintain homeostasis. The sympathetic division excites the heart to initiate increases in CO to meet the demand for higher blood circulation when the body is placed under physical stress, such as during physical activity. When the physical stress is removed, the parasympathetic division dominates to slow down the work rate of the heart (and subsequently the CO) in order to regain homeostasis (Meyer et al., 2002).

With repeated exposure to the physiological stress from training, the homeostatic perturbations to future stressors are reduced. This means that the body adapts physiologically after a suitable stressful episode to become resilient: it recovers more effectively from training loads to be in a state of readiness for the next bout. The interplay between the autonomic nervous system's regulation of the heart, physiological adaptations in response to training, the athlete's readiness to train and optimal performance has received much attention in recent sports orientated work. Specific focus has been on objective, non-invasive measures such as heart rate variability (HRV), HR recovery and HR acceleration in response to physical exertion (Bellenger et al., 2016).

Also, consumer orientated wearables, mobile applications and other portable devices have gained traction in the monitoring of the heart's regulation mechanism, providing a longitudinal window of insight into the ALS's fluctuating states of autonomic nervous system to gauge recovery and readiness to train (Chrismas et al., 2019; Dobbs et al., 2019). The bulk of evidence for the value of HRV in physiological monitoring are based on data from recreational and lifestyle athletes, with no, little or moderate training. However, the relationship between HRV and training loads in *elite* athletes remains open for interpretation (Plews et al., 2013).

Monitoring training loads in conjunction with the athlete's individual, physiological response may become a prime mechanism to manage recovery and avoid accumulated fatigue (Plews et al., 2013). This crossroad intersection of mobile, wearable technology and demand for healthier athletes may now accelerate the growth of research into recovery and overall 'readiness to perform'.

The HRV is a measure of the variability of the time between successive R-R intervals (also referred to as the inter-beat intervals) on an electrocardiogram (McArdle et al., 2010; Bellenger et al., 2016), often expressed as the root of the mean sum of squared differences, or the standard deviation, put as:

$$HRV = \sqrt{\frac{(x_i - \bar{x})^2}{n}} \tag{3.5}$$

where x_i represents the time for $R - R\ interval_i$, $\bar{x}$ is the mean or average of the time intervals and n the sample size of measurements. An important distinction must be made between parasympathetic tone and parasympathetic modulation: the R-R interval reflects the tone (regulation of the heartbeat), whereas the HRV reflects the *changes or modulation* in the tone as the heart reacts to demand (Plews et al., 2013). The R-R interval is a reflection of HR. Longer intervals produce a slower HR and shorter intervals a faster HR.

Higher HRV therefore implies the heart responds to stimuli and beats as per required physiological demand – the HR rhythm is therefore not constant but fluctuates. Low variability may be interpreted as shorter R-R intervals, not beating on demand, but at a more constant rate to maintain circulation. Higher variability is (mostly) associated in endurance athletes with a lower HR and parasympathetic dominance at rest (Sandercock et al., 2005); the heart is coping well with physiological demand and is not under stress. Low variability and a subsequent higher HR at rest are associated with sympathetic dominance (perhaps rather the withdrawal of the parasympathetic influence); a sign that the body might be under stress and/or struggling to recover (McArdle et al., 2010).

HRV may be a sensitive and accessible biomarker to monitor the parasympathetic modulation and positive (or negative) physiological adaptations in response to training loads (Bellenger et al., 2016; Dobbs et al., 2019). Perhaps more so, the day-to-day changes in HRV in relation to the average (or normal values) of the HRV is a biomarker for response to athletic training (Dobbs et al., 2019). A healthy balance between sympathetic and parasympathetic input to the heart exists when the HRV is large, a status that is achievable with regular physical activity (training) and the athlete is allowed proper recovery periods after training. Low variability, as a result of inadequate recovery but continuous physiological stress when the body is not ready to respond optimally, is a risk factor for the following: heart failure, myocardial infarction, sudden cardiac death (McArdle et al., 2010; Sandercock et al., 2005) as well as the Overtraining Syndrome (OTS) (Bellenger et al., 2016; Plews et al., 2013), discussed further in § 4.6 *The overtraining syndrome*.

3.1.4 Thermoregulation

Physical activity and exercise are disruptive to the athlete's thermal equilibrium (Nagashima et al., 2012; Mora-Rodriguez, 2012). A bidirectional deviation of 2°C from the optimal core temperature of 37.7°C is well tolerated through the regulatory systems, however disruption of bodily and the thermoregulatory systems occurs at a 3°C deviation. Serious and often irreversible damage may occur in the absence of intervention (Nagashima et al., 2012).

More than 75% of the mechanical energy generated by muscles is released as heat due to metabolic inefficiencies; dangerous build-up of this heat, which if not effectively dissipated out and away from the body, leads to disregulation of the core temperature (Wendt et al., 2007; McArdle et al., 2010). Thermoregulation of the athlete's body during activity is primarily directed to prevent and protect against overheating, that is, to counter heat stress (McArdle et al., 2010) in favour of thermal homeostasis. Thermoregulation mechanisms sustain the body's core temperature through carefully balanced heat exchanges that store some heat within a healthy range for optimal physiological function, despite environmental influences.

Heat exchange

Excess heat is dissipated when the body's core temperature rises; heat is generated when the core temperature declines. Heat is gained through the basal metabolic rate, muscular activities, hormones, some thermic effect of foods, postural changes. Heat is also absorbed from the environment through solar radiation, radiation and conduction from warm surfaces, and convection through warm gas or liquid. Excess heat is dissipated via radiation, convection, conduction and evaporation. (McArdle et al., 2010). To remain at a stable core temperature by storing heat, heat gain and heat dissipation is concurrent in the heat-balance equation (Wendt et al., 2007):

$$Hs \ = \pm R \pm Cv \pm Cd \pm -Ev \tag{3.6}$$

where Hs is heat storage, R is radiation, Cv is convection, Cd is conduction and Ev represents evaporation through sweat and pulmonary ventilation.

Note that Ev only removes heat: without sweating, thermoregulation becomes severely impaired when heat gain becomes excessive and cannot be fluxed through the other three mechanisms. The threshold where sweating becomes the main dissipation mechanism is at > 33°C dry-bulb temperature and > 60% relative humidity (Mora-Rodriguez, 2012). A hot and humid environment lowers both the temperature gradient as well as the water vapour pressure between skin and the air, thereby limiting both conduction of heat from the skin and the evaporation of sweat. The heat exchange between the athlete's body and the environment is hindered; often to such an extent that heat stress leads to some heat illness or injury (Wendt et al., 2007; McArdle et al., 2010), explained further in § 4.7 *Environmental stressors in running*.

Physiological strain and levers in thermoregulation

Internal and external factors that affect the amount of physiological strain during exercise in the heat are compiled from McArdle et al. (2010); Wendt et al. (2007); Nagashima et al. (2012); Mora-Rodriguez (2012) and listed here as:

- The environmental heat load: air temperature, relative humidity, convective warm air and/or liquid currents, heat transfer from conductive surfaces and radiant heat gain
- body size and composition
- training status (aerobic fitness)

- degree of acclimatisation
- intensity of physical activity
- clothing characteristics
- hydration and nutrition
- hormonal changes (such as during pregnancy)
- pre-exercise body temperature (modulated through pre-cooling)

El Helou et al. (2012) analysed 1 791 972 runners performances from six major marathons in the northern hemisphere ranging between 2001 and 2010, across four weather factors (air temperature, humidity, dew point, atmospheric pressure) and pollution levels. They found only air temperature to be significantly correlated with speed and withdrawal rates. Running speeds and withdrawal rates were linked with the differential from an optimal air temperature: speed would decrease and withdrawal rates increased when the temperature was above some optima. However, these environments did not qualify as a hot and humid environment, thereby the combined effect of temperature and humidity was not observed. Lee et al. (2010) showed a greater linear strength of association between decreased running speed and a rising core temperature in a hot and humid environment ($R^2 = 0.42$) when compared to other studies ($0.11 < R^2 < 0.15$) in cooler and less humid conditions.

Some of the factors mentioned are levers or interventions geared to develop heat tolerance and prevent heat injuries. Shorter term interventions include whole-body precooling, adequate hydration and appropriate clothing. Acclimatisation and aerobic fitness induce physiological adaptations that have longer lasting effects (Wendt et al., 2007; McArdle et al., 2010).

An aerobically fit individual have enhanced heat dissipation rates to compensate for higher metabolic heat production, a lower threshold to initiate sweating, a higher sweat rate and an improved VO_2-max in a temperate environment. At the same intensity (percentage of VO_2-max), aerobically fit individuals are able to tolerate more heat accumulation than an untrained individual can before the onset of fatigue (Mora-Rodriguez, 2012).

The physiological adaptations induced by aerobic fitness and acclimatisation are similar, although acclimatisation further enhances these modulations (Mora-Rodriguez, 2012). An effective CO from a stabilised CVS during physical exertions and augmented sweating mechanisms are the main results from acclimatisation through (Wendt et al., 2007; McArdle et al., 2010; Mora-Rodriguez, 2012):

- Decrease in HR during submaximal activity
- lower skin and core temperature at rest and during submaximal activity
- increased plasma volume
- decreased concentration of sodium and chloride in sweat (thereby also defending the plasma volume)
- increased sweat rate and sensitivity (the body has a lower threshold to initiate sweating)
- lower dependence on carbohydrate catabolism
- improved vasodilation to shunt warm blood to the skin (increased cutaneous blood flow)

These adaptations are complimentary to each other through their interaction. The main distinguishing feature of an acclimatised athlete is the ability to distribute the CO to simultaneously support active muscle for work and dissipate heat at the skin. With a lower core temperature at rest and the earlier onset of heat dissipation before an appreciable increase in core temperature, an athlete's time to voluntary exhaustion in the heat is prolonged (McArdle et al., 2010; Mora-Rodriguez, 2012; Nagashima et al., 2012). Yet, after acclimatisation of athletes at intensity of 60% VO_2-max for 9 to 12 consecutive days in 40°C, exhaustion remained when the core temperature of 40°C was reached. It is argued by some that there is a critical core temperature T_r at $39.5°C < T_r < 41.5°C$ at which hyperthermia becomes a crisis (McArdle et al., 2010; Nagashima et al., 2012), with cardiovascular and neurological failure passing 41.5°C (McArdle et al., 2010). Yet, there is evidence in Lee et al. (2010) to refute the critical core temperature, reporting from several studies on core temperatures above 39.5°C from asymptomatic runners during outdoor running in the heat. Normal thermoregulatory function was observed approximately 90 minutes after 15 minutes of running at 70% of the aerobic capacity. This may translate to a thermoregulatory homeostasis return time of about four to five times the duration of the exercise (Kenny and McGinn, 2017).

3.1.5 The neuromuscular system's role

Movement patterns are learnt activities and instantiate itself after repetitive actions to acquire a skill. Movement patterns are subject to the neuromuscular system, controlling the activities in the MSS such that movements may be balanced and coordinated for the athlete to move effectively and efficiently (Enoka and Duchateau, 2019). The interactive unit between a nerve and muscle is referred to as the motor unit. Muscle action is initiated and sustained by the firing of the motor units and relaxed when firing stops, a process that is governed by the complex neurological system (Chleboun, 2005).

In the MSS the sequencing of firing motor units is profoundly linked to the adaptation of the ALS to the sports tasks and environment. Poor neuromuscular control (i.e. uncontrolled or wrong sequencing of motor unit firing) must be corrected during normal training and rehabilitation (after an injury) through motor retraining (Brukner and Khan, 2006). The elongation of muscles with a subsequent contraction to shorten the muscle is known as the stretch reflex. The stretch reflex is responsible for generating concentric contraction in the muscle when the muscle is elongated, through motor unit firing in response to a stretch and is facilitated by the central nervous system to stabilise joints (Chleboun, 2005).

3.2 Fitness and health: towards a well prepared athlete

There is a simple, anonymous quote "athletes eat and train, they do not diet and exercise". A sharp contrast in the wording of this phrase must be noted: dieting and exercise are related to restricted intake of energy and increased output of energy in order to loose or maintain weight. This practice is far removed from the athlete's ideology – take on board what is needed to provide and restore energy and condition the body to perform optimally.

Fitness is the complete match of the athlete to their environment, encompassing physiological, psychological, sociological and theological matching (Meyer et al., 2002). However, fitness and health may be defined separately (Maffetone and Laursen, 2016, p.1):

> *"Fitness describes the ability to perform a given exercise task, and health explains a state of complete, mental, social, and physical well-being, where all bodily systems (nervous, hormonal, immune, digestive, etc.) function in harmony."*

From this definition, an athlete may be fit enough to perform a task, yet in an unhealthy state when the subsystems are functioning sub-optimally. An example being an athlete running a well-deserved time in a race, but carries the signs of MSS dysfunction on their body with strapped-up knees, muscle cramps or limping closer to the end of the race. The athlete is still *able* to perform the running task, but not in an optimal healthy state.

The physical training regime must therefore imbue the above-mentioned components in the ALS (Meyer et al., 2002; Maffetone and Laursen, 2016), through well-planned training intensity, volume and nutritional intake. Maffetone and Laursen (2016) argue that excessively high training loads (in both volume and intensity) achieve the opposite of its original intention: a fatigued and unhealthy athlete. Poor health is evident in athletes with the 'no-pain-no-gain' mindset: extending far beyond their developed physical capacity for training, without allowing the body to sufficiently dissipate the stress from training and adapt to the demand (Maffetone and Laursen, 2016).

3.3 Training: a function of biomechanics, physiology and the neuromuscular pathways

In this thesis, the term 'training' encompasses all activities related to strengthening the subsystems in complex interactive processes and is a synergy between exercise (physical and physiological overloading) and recovery (to repair and build structure). At the core of any training regime lie augmented muscle strength, endurance, and power production to reduce injury risks and promote the health of any athlete and/or individual (Brumitt and Cuddeford, 2015). Training activities (should, if done correctly) culminate in a physically resilient and fit athlete (Brukner and Khan, 2006; McArdle et al., 2010; Maffetone and Laursen, 2016).

3.3.1 Adaptation through micro-trauma

Positive responses to kinetics require healthy kinematic traits of any athlete, achievable by placing the body under physical stress in order to stimulate physiological processes that systemically strengthen the subsystems of the ALS, thereby adhering to the overload principle (Flynn et al., 2019; Enoka and Duchateau, 2019). Within the context of exercise, stress implies some exertion above normal functioning, inducing physiological reaction in the CVS, MSS, and neuromuscular-, metabolic- and thermoregulatory systems for tissues to adapt to the overload of stress. The progressive adaptations allow higher stress loads to be applied to stimulate further improvements, thereby augmenting fitness and health (Flynn et al., 2019). The term exercise-induced muscle damage is associated with muscular and soft tissue growth in response to exercise training. The micro-trauma caused by mechanical stress overload of structures induces the inflammatory response, followed by physiological activities during the adaptation phase for muscles to grow and tissues to remodel (Fatouros and Jamurtas, 2016). As training and exposure to mechanical stress progress with time, muscles become more resistant to damage imposed by stress and if damaged, repair occurs at a faster rate. Small inflictions of stress will produce favourable adaptations (Clarkson and Tremblay, 1985).

Table 3.2 lists four main training methods, each targeting a subsystem or a complex of subsystems to build resilience (Brukner and Khan, 2006; Flynn et al., 2019). Although the CVS is only directly related to aerobic and anaerobic training, it is a crucial facilitator of the other training methods since all the body tissues involved depend on blood supply (Meyer et al., 2002; McArdle et al., 2010).

Table 3.2: Training the subsystems

Training method	Main target subsystem(s)
Endurance or aerobic	Cardiovascular and muscles
Anaerobic	Cardiovascular and muscles
Strength and power	Musculoskeletal
Flexibility, speed and agility	Musculoskeletal and neuromuscular pathways
Skills and cross-training	Musculoskeletal and neuromuscular pathways

3.3.2 Aerobic (or endurance) and anaerobic training

These training modalities strengthen the ability of tissues to optimise the two metabolic pathways: with oxygen supply (aerobic) and in the absence of oxygen (anaerobic) (Brukner and Khan, 2006). Endurance is defined as *"the ability to train and compete over longer time frames, with less fatigue and at faster paces"* (Maffetone and Laursen, 2016, p:3). Endurance training is therefore sometimes used as synonymous with aerobic training to sustain the ALS during extended periods of physical exertion.

An athlete augments their aerobic capacity, measured as the VO_2-max ($\frac{ml\ O_2}{kg \cdot min}$) and becomes more efficient and effective in utilising glycogen stores in muscles through the aerobic pathway (Brukner and Khan, 2006; McArdle et al., 2010). There is a multitude of methods to predict VO_2-max. Direct VO_2-max assessment may be completed in breath-by-breath gas analysis during graded exertion protocols (Peric and Nikolovski, 2017; Brukner and Khan, 2006) in laboratories or indirectly predicted in either laboratories or real-world environments during walk/run tests (Mayorga-Vega et al., 2016; Brukner and Khan, 2006).

Sophisticated laboratory equipment that measure oxygen and CO_2 concentrations in an athlete's expired air is not easily accessible by recreational and lifestyle athletes (Brukner and Khan, 2006). Indirect predictions of VO_2-max on stationary bikes or motorised treadmills include (Peric and Nikolovski, 2017):

$$\begin{aligned}
\text{Bruce test} : VO_2\ max &= 14.8 - (1.379T) + (0.451T^2) - (0.012T^3) \\
\text{Balke test} : VO_2\ max &= 1.444T + 14.99 \\
\text{Astrand-Ryhming test} : VO_2\ max &= 100 \times \frac{(0.00212W + 0.299)}{(0.769\mathrm{HR}_{\max} - 48.5)} \\
\text{PWC test: } VO_2\ max &= 3.5 + 12W_{end}
\end{aligned} \tag{3.7}$$

where T is total time in minutes, W is maximal workload in Watt's, and W_{end} is the maximal workload divided by body weight in kilogram, expressed as $Watt.kg^{-1}$.

Outside the laboratory, methods to predict VO_2-max include walk/run tests of various lengths (distance or time defined), ranging between 600 meters to 5 km, or 4 minutes to 15 minutes (Mayorga-Vega et al., 2016), The predicted VO_2-max is more accessible, requiring only HR, workload (time taken to complete a set distance), weight, and gender, shown in equation form as (McArdle et al., 2010):

$$\begin{aligned}
VO_2\ max = 132.853 &- 0.0769W - 0.3877A \\
&+ 6.315G - 3.2649T_f - 0.01565HR
\end{aligned} \tag{3.8}$$

where W is weight in pounds, A is age, G is binary (1 for male, 0 for female), HR is heart rate and T_f is the time to finish a 1.6 km track run or walk. Wearable technologies, such as RWs, probably use HR readings during a set time window during which the athlete runs to determine the VO_2-max.

Aerobic training profits the muscle by increasing the number of mitochondrial cells (i.e. the energy production unit), glycogen storage, its ability to utilise free fatty acids and in vascularity (Brukner and Khan, 2006; McArdle et al., 2010). Blood flow to skeletal muscle during strenuous physical activity may increase 10-fold in young, healthy individuals (Betts et al., 2013), with McArdle et al. (2010) reporting up to a 21-fold increase. This results from a five-fold increase in total CO, from the general 5 L at rest (with 20% distributed to skeletal muscle) up to 25 L, with more than 84% distributed to the skeletal muscle (now a total of 21 L). The CVS response in the MSS to this increased demand of circulation is arteriogenesis (enlargement of arterial cross-sectional area), angiogenesis (formation of new blood vessels), and the increase in the capillarisation surrounding the muscle. This increase in the capillary-to-muscle fibre ratio is a reflection of the beneficial effect of endurance training. The surface area between muscles and blood for nutrient and metabolic gas exchange is enlarged, providing for a higher rate of energy production (McArdle et al., 2010). Together these adaptations in circulation and tissue neo-genesis improve the vascularity of the MSS.

Through aerobic training the CO (in ml/min) from the heart is increased, providing for higher blood volume circulated through the body. The heart, as a muscular pump, becomes stronger as it can pump more blood per beat through the body (i.e. an increased stroke volume), thereby acquiring a lower HR and reduced blood pressure (Brukner and Khan, 2006; McArdle et al., 2010). Cardiac output is a function of the stroke volume and HR, in Equation 3.9:

$$CO = SV \times HR \tag{3.9}$$

Additional CO in response to physical demand for increased circulation is obtained through increasing HR, stroke volume or both. In athletes, an increased stroke volume is the main mechanism by which circulatory demand is met (McArdle et al., 2010).

It is through aerobic training that the heart functionally adapts to match the circulatory demand from physical activity. Aerobic training places a volume load on the heart, to which it responds by increasing the stroke volume through cardiac adaptations. Stroke volume is enhanced through stronger contractions of the myocardium and a higher blood volume in the ventricle. The heart adapts as follows to obtain a larger stroke volume (McArdle et al., 2010; Meyer et al., 2002; Bellenger et al., 2016):

- Increased mass and volume of the left ventricle (through training-induced plasma volume expansion). Myocardial eccentric hypertrophy increases the size of the left ventricular cavity, and concentric hypertrophy modestly thickens the walls of the ventricle, thereby allowing for a stronger ventricular systole and more blood is ejected from the heart per beat. An augmented stroke volume through stronger heart muscle furthermore follows the same physics laws as the for MSS stress-strain behaviour from § 3.1.1. The tension-length relationship of cardiac fibres is known as Starling's law: tension develops in the cardiac muscle when placed under a volume load, that is, elastic energy is stored. A stronger contraction of the muscle fibres results from the release of the stored elastic energy. The strength of the contraction is therefore directly proportional to the length of the fibres.

- Reduced stiffness (or improved flexibility) in the myocardium and arterials (allows for expansion of the heart's ventricular walls to increase the volume inside the ventricle)

- Increased filling time during ventricular diastole through a lowered HR

- Improved intrinsic contractility of the myocardium

Since the stroke volume is affected by the time allowed to fill during diastole, HRV is an indication of the heart's autonomic modulation. Contraction (systole) is initiated through sympathetic stimulation, and relaxation (diastole) through parasympathetic tone. A prolonged diastole, that is the time between consecutive beats or systolic contraction of the ventricle, allows more blood to fill the chamber and load the heart muscle eccentrically (consequent build-up of elastic energy). In this way, HRV provides some eluded insight into the heart's autonomic balance to produce the required CO through an augmented stroke volume from a stronger heart muscle.

With a larger stroke volume, CO at rest or sub-maximal exercise may be maintained at a lowered HR, reducing the metabolic load on the heart. For some, an increased parasympathetic modulation is seen through a decreased resting HR and an increased HRV (Bellenger et al., 2016), both indications of a healthy, well-adapted heart. The heart is seen to be in a state of 'readiness' to do intensive work as required by training, although this might not always be the case. Athletes have unique autonomic (and HRV) footprints (Plews et al., 2013). In Plews et al. (2013) it is shown that the function between the R-R interval (on the x-axis as a reflection of parasympathetic tone) and the $\log(rMSSD)$ (on the y-axis, reflecting parasympathetic modulation) is bell-curved. That is, HRV increases and parasympathetic modulation is maximised at some HR (or R-R interval), after which modulation is saturated and HRV decreases again. HRV must not be an isolated, linear predictor of training and/or performance readiness. A shortened diastole is not necessarily indicative of higher sympathetic tone, but may be the withdrawal or lowered parasympathetic tone (Bellenger et al., 2016). A large HRV may be masking some other autonomic imbalance, such as insensitivity to sympathetic stimulation (Plews et al., 2013). Furthermore, the review by Bellenger et al. (2016) reports that in some studies there were concurrent increases in both HRV and hr recovery, with others showing decreases in HR acceleration after overload training. The reduced HR acceleration might be a marker for training-induced fatigue, as the heart does not respond to sympathetic input or is slow to respond. The findings are equivocal to the autonomic balance following training in the absence of other contextualising variables.

HRV, or any measure to quantify the autonomic regulation of the heart, as with all other physiological markers, is best approached and used within a ST framework: consider the interactions of all the units that make up the ALS to identify a fatigued athlete, and not just a linear assumption that the sum of a high HRV and a low HR equates to training or performance readiness.

Effective aerobic training has been defined in literature in terms of training intensity, marked out within zones of physiological measures such as VO_2-max, lactate acid markers, ventilatory threshold, HR-zones, rate of perceived exhaustion and metabolic equivalents amongst others (McArdle et al., 2010; Seiler and Kjerland, 2006; Sylta et al., 2014). Lactate acid zones are defined in Neal et al. (2011): zone 1 is below the threshold, between the threshold and the lactic acid turn point constitute zone 2 and zone 3 is marked as above turn point. Seiler and Kjerland (2006) found evidence amongst young cross-country skiers that aerobic training is performed mostly under the lactate acid threshold (determined for the group of skiers as below 81% of HR-max). Neal et al. (2011) conclude from others' experimental work (Ingham et al., 2008; Esteve-Lanao et al., 2007) that aerobic training effect (through physiological adaptation) is greatest when training in zone 1 amounts to 80% of training time and 20% or less in zones 2 and 3. In their own analysis of triathletes, Neal et al. (2011) concur these findings when only modest to negligible adaptations where found in triathletes who spent <80% of their training in zone 1.

Measuring the lactate concentration is not a practical approach to monitor time spent in training zones. With HR, being much more accessible, training intensity in the field may be monitored through time spent in HR-zones that correspond to the lactate acid thresholds (Neal et al., 2011). Sylta et al. (2014) matched the lactate threshold zones to HR-zones. Zone 1 (low-intensity) corresponds to 55% to 82% of HR-max; zone 2 between 82% and 87% and zone

3 is bounded between 92% and 97% of HR-max. Various HR-zones have been suggested in literature where an aerobic effect may be obtained (McArdle et al., 2010; Ignaszewski et al., 2017; Khalil and Sornanathan, 2010; Sylta et al., 2014; Maffetone, 2015). Depending on the desired aerobic effect, an athlete spends a certain amount of time within these zones. The cut-off points for the zones are not conclusive in literature, although early work by Martti Karvonen has made significant contributions to define the effective zones (Ignaszewski et al., 2017). Training intensity zones as developed by Martti Karvonen are a function of maximum HR, the HR-reserve, age and gender.

The HR-zones are calculated as shown in McArdle et al. (2010), with the 180-formula given by Maffetone (2015) in Equation 3.10:

$$
\begin{aligned}
HR_{max} &= 208 - 0.7 \times Age \\
HR_{UL} &= (HR_{max} - HR_{rest}) + K_u \times HR_{rest} \\
HR_{LL} &= (HR_{max} - HR_{rest}) + K_l \times HR_{rest} \\
HR_{180} &= 180 - Age - \text{ recovery } - \text{ other conditions } + \text{ No problem}
\end{aligned}
\tag{3.10}
$$

The HR_{max} is predicted using the updated formula from the traditional $220 - Age$ formula (McArdle et al., 2010). The upper and lower HR limits (HR_{UL} and HR_{LL} respectively) are calculated using the Karvonen-method in McArdle et al. (2010) and Ignaszewski et al. (2017), with the HR reserve as $HR_{max} - HR_{rest}$. K is the constant used to calculate the thresholds (or cut-off points to define the HR zones) at which training should be completed to gain an aerobic effect. These zones differ in literature. Ignaszewski et al. (2017) indicate that Karvonen defined the zones for $K = \{0.3, 0.4, 0.6, 0.9\}$ while McArdle et al. (2010) uses $K_u = 0.85$ and $K_l = 0.5$ as the aerobic effective zone. Khalil and Sornanathan (2010) subset more zones with cut-off points at $K = \{0.65, 0.75, 0.8, 0.85, 0.9\}$ with disparate intensity assigned to each zone.

The 180-formula works as follows: subtract 10 if the athlete is recovering from an illness or injury (zero otherwise); subtract another five if the athlete is injured, has chronic underlying conditions, allergies or are on chronic medication (zero otherwise); add five if they have been training without any of the previous two constraints for two years (zero otherwise) (Maffetone, 2015).

High intensity interval training facilitates anaerobic training and is characterised by short bursts of high intensity work, followed by a period of recovery. During the high intensity period, the body utilises the anaerobic pathway to provide the energy required for the surges in power (Buchheit and Laursen, 2013). The body's tolerance to lactic acidosis, (developed from the presence of lactic acid in the muscle after the quick glucose metabolism), is improved. Significant improvements of VO_2-max when running at the blood lactate threshold were obtained from well-trained distance runners after a high intensity interval training program that involved both level and uphill running(Ferley et al., 2014). Recovery after intense bouts of activity is allowed to dissipate the lactic acid from the muscles. Progressively the body's ability to remove lactic acid is enhanced and muscles can generate more power (Brukner and Khan, 2006).

3.3.3 Strength, power, flexibility, speed, agility, cross-training and skills

Strength, power, flexibility, speed and agility training are the drivers behind adaptations in the mechanical properties of the tissues in the MSS, leading to changes in the stress-strain relationships discussed earlier in § 3.1.1 *The stress-strain curve*.

Strength and conditioning improves the capacity of the ALS to attenuate load and should form an important component in the training program of runners, both pre-season and during the season (Barton, 2018). Strength is the maximum force that a muscle is capable to produce and power is the maximum amount of work that a muscle can do. Muscle contraction (concentric and eccentric) under progressive loading stimulates cellular changes and muscle growth, enabling higher strength and power production (Brukner and Khan, 2006). In the presence of adequate stimuli (the 'overload' principle), the neuromuscular system and MSS interact by augmenting the neural drive and contractile properties of the muscle (Enoka and Duchateau, 2019), with the sequential firing of motor units in reaction to the extra load placed on the muscle.

During strength training, the stress-strain curve (Figure 3.3) moves upward, with an increase in elasticity (a steeper slope in the linear region); subsequently more stress may be absorbed during plastic deformation. Failure will still occur at the same strain point as in a weaker muscle, although a higher force (stress at σ_2) is required to reach this length of deformation (Chleboun, 2005). The muscle has therefore adapted to be more resilient in the face of increasing forces.

Flexibility is a characteristic of good physical health and important for runners as it allows for the required joint range of motion to generate torque through muscular action and elastic return (Nikolaidis et al., 2018, 2019). Increased range of motion results in longer moment arms through which torque is produced. It is important to develop muscle strength in the newly acquired range to stabilise the movement (Brukner and Khan, 2006). Agility improves reaction times and speed, a combination of coordination and strength (Brukner and Khan, 2006; Shivalingaiah et al., 2016). An agile athlete is more likely able to also display good dynamic balance, rhythm and spatial awareness, important determinants for performance and protects against injury (Shivalingaiah et al., 2016). Cross-training is useful to reduce the amount of stress that must be absorbed in weight-bearing activities and yet maintain aerobic fitness (Brukner and Khan, 2006). Skills are a direct result of reinforcement of favourable neuromuscular pathways and muscle sequencing, and are highly dependent on an intact MSS (Barton et al., 2016).

3.3.4 Training dosage: volume, frequency, intensity, rest, and periodisation

Although adequate overload is required to stimulate adaptations in the ALS, the training load dosage must incorporate a balance in volume, frequency, and intensity. Recovery (rest) is required to produce the training effect. The frequency of training (number of training bouts per time-period) must therefore allow for adequate rest periods (Brukner and Khan, 2006). Volume refers to the quantity or duration of training (in distance per time period, or cumulative distance over a time period, or time spent running per time period), whereas intensity reflects the quality of training. Intensity is expressed as volume divided by duration, therefore variables such as running pace (min/km) is often used to report intensity of training (Nielsen et al., 2012). Either may be increased, although it is advisable that it is not done concurrently, but that volume precedes intensity (Brukner and Khan, 2006; Kreher and Schwartz, 2012).

Periodisation divides the training period into three distinct phases: conditioning, precompetition (or transitional), and competition. Aerobic and anaerobic fitness, strength and power are built during the conditioning phase. Techniques are honed during the transition phase together with continued conditioning. The focus shifts to competitive performance during the competition phase whilst maintaining basic conditioning. Training programs must allow optimal periodisation for athletes to peak during competition time (Brukner and Khan, 2006).

3.4 Running economy

The energy demand at a given submaximal velocity, measured as the steady-state oxygen consumption ($\dot{V}O_2$), is defined as the RE. Accounting for BW, RE is expressed in $ml.kg^{-1}.min^{-1}$. A lower value for the same running velocity indicates a better RE (Saunders et al., 2004; Barnes and Kilding, 2015). The RE varies across athletes with similar $V0_2$-max values. At 14 km.hr^{-1}, mean (range) for RE and $V0_2$-max were reported for male and female recreational runners at RE = 47.4 (46.0-49.5), $V0_2$-max = 54.2 (51.0-57.8) and RE = 47.3 (40.1-51.9), $V0_2$-max = 49.7 (45.2-54.1) respectively. At the same speed, elite runners measured RE = 39.9 (36.1-44.5), $V0_2$-max = 75.4 (68.2-84.1) and RE = 41.9 (38.7-46.9), $V0_2$-max = 66.2 (61.1-74.2) for male and female athletes. Although the $V0_2$-max is substantially higher for elite athletes than for recreational runners, the good RE values are better predictors of endurance performance. The elite athletes had more favourable RE at the same speed than the recreational athletes. That is, they use less energy to deliver the same performance (Barnes and Kilding, 2015).

Running economy is influenced by many factors, including training, the environment, physiology, biomechanics, anthropology, genetics, metabolic efficiency, cardiorespiratory efficiency, and neuromuscular efficiency (Saunders et al., 2004; Barnes and Kilding, 2015; Moore, 2016). Supporting BW and generating forward propulsion accounts for 80% of metabolic costs of running, followed by leg swing (7%) and lateral balance (2%), leaving 11% as unexplained (assigned to the costs of braking, ventilation and cardiac work) (Moore, 2016). It therefore makes sense to modify the biomechanical synergies required for BW support and forward propulsion in order to lower the RE. Through modification of biomechanics, a runner may improve their RE, although what may be economical for one runner may not hold for another runner (Barnes and Kilding, 2015). Moore (2016) found the following biomechanical adaptations to be beneficial for RE:

- a preferred stride length range (up to 3% shorter than the preferred length)
- lower VO
- larger stride angles
- greater leg stiffness
- lower inertia of the lower limb
- decreased leg extension at toe-off
- improved alignment with GRFs and leg axis during propulsion
- maintaining arm swing

Runners may become more economical over time, improving their RE as a result of high training volumes and years of running experience (Barnes and Kilding, 2015; Moore, 2016). Training to address biomechanical adaptations depends on the strength of the stimulus and the experience of the runner (Moore, 2016). As a result of run training, the neuromuscular system improves the recruitment of motor units, thereby decreasing wasteful movements and optimising specific movement patterns that are associated with efficiency to execute the run task (Barnes and Kilding, 2015).

Through strength training, the muscles' elasticity improves and energy lost during breaking decreases. Heavy-weight strength training in combination with endurance training was associated with augmented RE of well-trained triathletes. A significant 11% reduction in submaximal $\dot{V}O_2$ was reported for the intervention group after 14 weeks of heavy weight training, when compared with the endurance-only group (Saunders et al., 2004). Muscle and connective tissue elasticity are considered significant influencers of RE (Saunders et al., 2004; Moore, 2016). To account for the elasticity, it has been suggested that $\dot{V}O_2$ be proportional to the 0.66 or 0.75 power of BW, that is to say $RE \propto BW^{0.75}$ (Saunders et al., 2004). Plyometric training, or explosive strength training, enhances neuromuscular function through improved recycling of elastic energy, which translates into lower RE. Furthermore, acclimatisation (through training in the heat) improves RE by reducing the energy required to deal with heat stress (Saunders et al., 2004).

Environmental factors that influence RE include air resistance in outdoor running, altitude, the running surface, shoe-surface interactions, and orthotics. Firm, compliant shoe-surface interactions are beneficial to RE, so are barefoot or lightweight shoes (< 400 gram). Evidence for orthotics' effect on RE is conflicting (Saunders et al., 2004; Moore, 2016).

Therefore, many methods by which a runner may improve their RE exist, although individual responses to methods differ due to the various levels of adaptations in the MSS, the CVS, and the neuromuscular system.

3.5 Conclusion

Figure 3.5 provides a diagrammatic overview of the chapter.

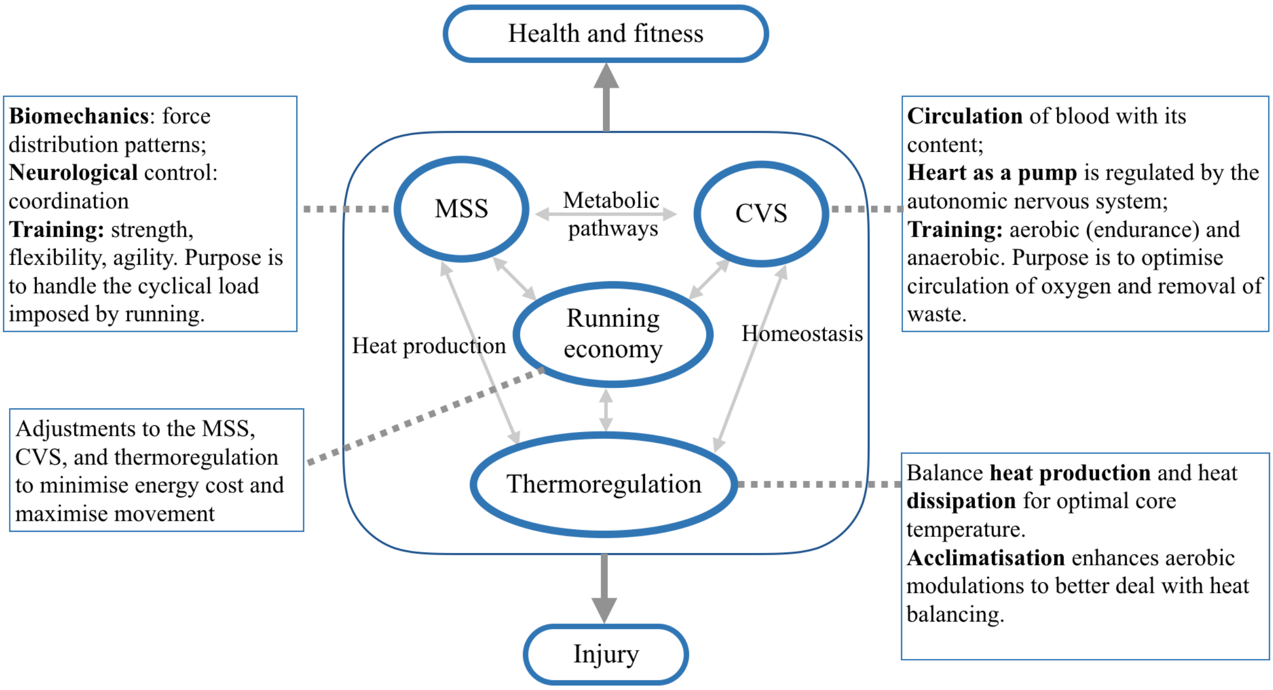


Figure 3.5: Most important aspects of the ALS

This chapter unpacked the most important subsystems of the ALS in its environment. The ALS moves when the MSS does positive work to displace the body through time and space, whereas the CVS does work to transport fuel, oxygen and waste to their intended destinations. The CVS may be thought of as the enabling network on which the MSS depends to do work. Heat released by muscular actions and metabolic activities during physical exercise is dissipated out and away from the body through the thermoregulatory mechanisms in order to maintain thermohomeostasis. Sweating becomes the most effective heat dissipating mechanism for the athlete when the core temperature starts to rise from higher internal heat production by active muscles. Degrading processes, in some cases fatal, follow when the thermoregulatory system fails.

Training benefits all the subsystems of the ALS. Exercising in the heat can be leveraged by the athlete for improved conditioning; however, care must be taken as not to overexert the body's physiological capacities to avoid unwanted consequences such as tissue damage or heart failure. The RE is a complex, multifactorial measure whereby the ALS adjusts biomechanical gait parameters, heat dissipation mechanisms, and circulatory requirements to minimise energy cost of running whilst optimising movement patterns. A fit athlete is not necessarily a healthy athlete: the balance between overload and recovery must be maintained to produce desirable physiological modulations in the athlete.

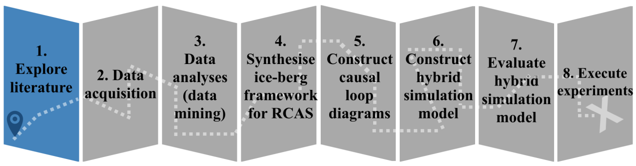
Whereas this chapter dealt with how the subsytems *should* function, the subsequent chapter describes what happens when the subsystems are exerted beyond their physiological and biomechanical capacities: injuries.

Chapter 4

When the locomotion synergy goes wrong: risk factors and injuries

Contents

This chapter formulates part of task 1 from the research methodology.



This chapter relates overuse injuries, specifically RROIs, to risk factors and environmental influences. Some domain specific solutions to injuries are discussed. Recently, the term *gradual onset* injuries have been used instead of *overuse*, however in order to build on the momentum regarding ST and running related injuries in Hulme et al. (2017b) and Hulme et al. (2018), the thesis remains with *overuse*.

4.1 Injury prevalence

Injuries are unwanted side effects from sport participation (Malm et al., 2019; Maffetone and Laursen, 2016), yet the prevalence remains despite extensive research to identify root causes, prevent injuries, and find effective treatments for the injured athlete (Barton et al., 2016). Literature shows varying incidence of RROIs:

- Earlier between 1978 and 1980, 1819 injuries were reported by 1650 surveyed runners with patellofemoral (knee) pain being reported the most (Olney, 2005).

- Among studies with follow-up periods ranging between one week and 18 months, RROI incidence ranged between 19% and 78% (Barton et al., 2016).
- Epidemiological studies reported between 27% and 70% incidence of RROIs amongst recreational and competitive distance runners within a one-year period, with 80% of injuries involving the knee or joints below the knee (Ferber et al., 2009).
- Rates between 2.5 and 12.1 injuries per 1000 running hours with 30% to 70% of injuries requiring reduction in training and 70% requiring medical attention were reported by Gijon-Nogueron and Fernandez-Villarejo (2015).

The high incidence range is perhaps a reflection of the complexity behind reporting and classifying injuries. In addition, Kluitenberg et al. (2015) report that injury prevalence is different among populations of runners (track runners, novice runners, recreational runners, cross-country runners, long-distance runners, marathon runners, and ultra-marathon runners). There is also evidence for a U-shaped pattern between the time-loss due to injury and the running distance, where ultra-marathon and sprinting runners presented with the highest proportions of injuries that involved time-loss (Kluitenberg et al., 2015).

4.2 Typical running related overuse injuries

In the MSS, ligaments are sprained, muscles are strained and bones fracture when the tissue fails along the stress-strain curve during plastic deformation (refer to Figure 3.3). Soft tissue injuries are graded depending on the amount of fibre separation, which coincides with failure within in the plastic region. Bones are first strained, advancing to small cracks, and finally a stress fracture develops under progressive overuse. A full rupture of soft tissue or fracture of bone occurs at the failure point, and can be a result of long-standing overuse or a sudden, traumatic event such as a collision or a fall (Brukner and Khan, 2006).

The typical aetiology[1] for RROIs involves the repetitive loading of structures without adequate recovery to repair the strained tissue, creating an imbalance between load and restitution of tissue (Larsen et al., 2016). Put otherwise, structures are loaded while they have not yet returned to a fully functional state. The repetitive forces create an additive effect leading to micro-trauma, setting the inflammatory process in motion with swelling following suit. The musculotendinous unit is most affected by overuse. The unit becomes fatigued with repetitive, sub-maximal loading, especially under rapidly applied tension while in a stretched position (Herring and Nilson, 1987). Recently, Gabbet (2016) stated that overuse injuries are the result of a training load error, whereby an athlete is training too hard too soon or not enough to protect against micro-failures. In this way, training becomes a contradictory activity; whereby imbalanced (uncontrolled volume, intensity and duration) training might instigate an injury and balanced training puts forth a resilient and prepared athlete.

Figure 4.1 plots the common RROIs onto a human body chart, collected from Brukner and Khan (2006); Larsen et al. (2016); van der Worp et al. (2015); Wen et al. (1997); Diebal et al. (2011); Louw and Deary (2014); Ribeiro et al. (2011); Via et al. (2018); Dixit et al. (2007); Via et al. (2016); Astur et al. (2015); Kahanov et al. (2015); Reshef and Guelich (2012); Pujalte and Silvis (2014); and Arnold and Moody (2018).

The typical RROIs are explained from left to right in Figure 4.1. *Stress fractures* are microfractures (small bone breakages due to the imbalance between osteoblast and osteaclast activity) brought on by repetitive loading below the single cycle failure threshold (Brukner and Khan, 2006; Astur et al., 2015). These occur in the foot (calcaneous[2], talus, metatarsals

[1] causation, set of causes
[2] heel bone

and tarsal bones), lower limbs (femur[3], fibula and tibia[4]), the pelvis (pubic bone and sacrum), the lumbar vertebra and the scapula[5] (when running with weights in the hands) (Brukner and Khan, 2006; Kahanov et al., 2015). *Tendinopathy* (of the patella[6], hamstring, achilles and other muscles) results from tendinosis and is marked by collagen disarray, fibre separation, neovascularisation[7], loss in parallel orientation of fibres and decreases in fibre diameter of the involved tendon. The cellular and fibril changes alter the mechanical properties and strain capacity of the tendon (Brukner and Khan, 2006; Via et al., 2016).

Runner's knee (also patellofemoral[8] pain) is a collective term for pain in and around the patella, in the absence of other pathologies. Pain is caused by maltracking of the patella during knee motion, due to weak and/or tight *quadriceps*[9], tight lateral structures and patellar hyper- or hypomobility (Brukner and Khan, 2006; Dixit et al., 2007). Synonyms used are patellofemoral joint syndrome, anterior knee pain and *chondromalacia patellae* (Brukner and Khan, 2006). *Shin splints*, the colloquial term for *medial tibial traction periostitis* or *medial tibial stress syndrome*, present with diffuse pain along the medial border of the tibia. Causes have been listed as the excessive traction exerted on the periosteum[10] of the bone by tight calf muscles, inflammatory response of the periosteum and a stress reaction of the tibial bone (Brukner and Khan, 2006; Reshef and Guelich, 2012).

Pelvic bone overload, more specifically *pubic bone overload*, occurs when repetitive, mechanical stress is placed on the pubic bone with subsequent strain on tissues, resulting in failure of the local tissue when strained beyond the failure point. Failure may occur in the muscles, tendons, the bone or a combination of these. The pubic bone is overloaded by the immoderate forces created from increases in tone of the *rectus abdominus* and *adductor* muscles, lumbopelvic[11] instability, sacroiliac[12] joint dysfunction, limited hip range of motion and shortened hip flexors leading to pubic bone stress (*osteitis pubis*) or a stress fracture of the pubic bone (Brukner and Khan, 2006; Via et al., 2018). *Sacroiliac joint dysfunction* is a collective term to describe any abnormality in the biomechanics and mobility (hyper- or hypo-mobility) of the joint (Brukner and Khan, 2006). Connecting the pelvis and the spine, this joint is important for force transfer from the lower limbs to the trunk during stance, walking and running. Hyper- or hypo-mobility (stiffness) of the joint greatly influences gait by creating imbalances in the pelvis and spine, resulting in overload of some structures and inactivity of others (Brukner and Khan, 2006; Dalton, 2005). Sacroiliac dysfunction contributes to pain and disorder in the groin (through pubic overload), buttock, hamstring and lumbar region (lower back) (Brukner and Khan, 2006; Ou-Yang et al., 2017). *Plantar fasciitis* is tendinosis of the *plantar fascia*[13] marked by collagen disarray with absence of inflammatory cells and occurs at the attachment to the calcaneus (Brukner and Khan, 2006). As a result of overload, microtrauma occurs in the plantar aponeurosis due to repetitive stretching stresses, causing damage to connective tissue (Ribeiro et al., 2011).

Compartment syndrome, or *chronic exertional compartment syndrome*, is defined as increased intramuscular pressure within a bounded fibro-osseous space, marked by a reduction in blood flow, tissue perfusion and muscular function. The compromised perfusion results in

[3]thigh bone
[4]shin bone
[5]shoulder blade
[6]knee cap
[7]formation of new blood vessels
[8]where the knee cap and thigh bone form a joint
[9]front muscle of the thigh
[10]membrane surrounding the bone
[11]the connection between the lower back and the pelvis
[12]joint between the wings of the pelvis and the sacrum
[13]fibrous band at bottom of foot

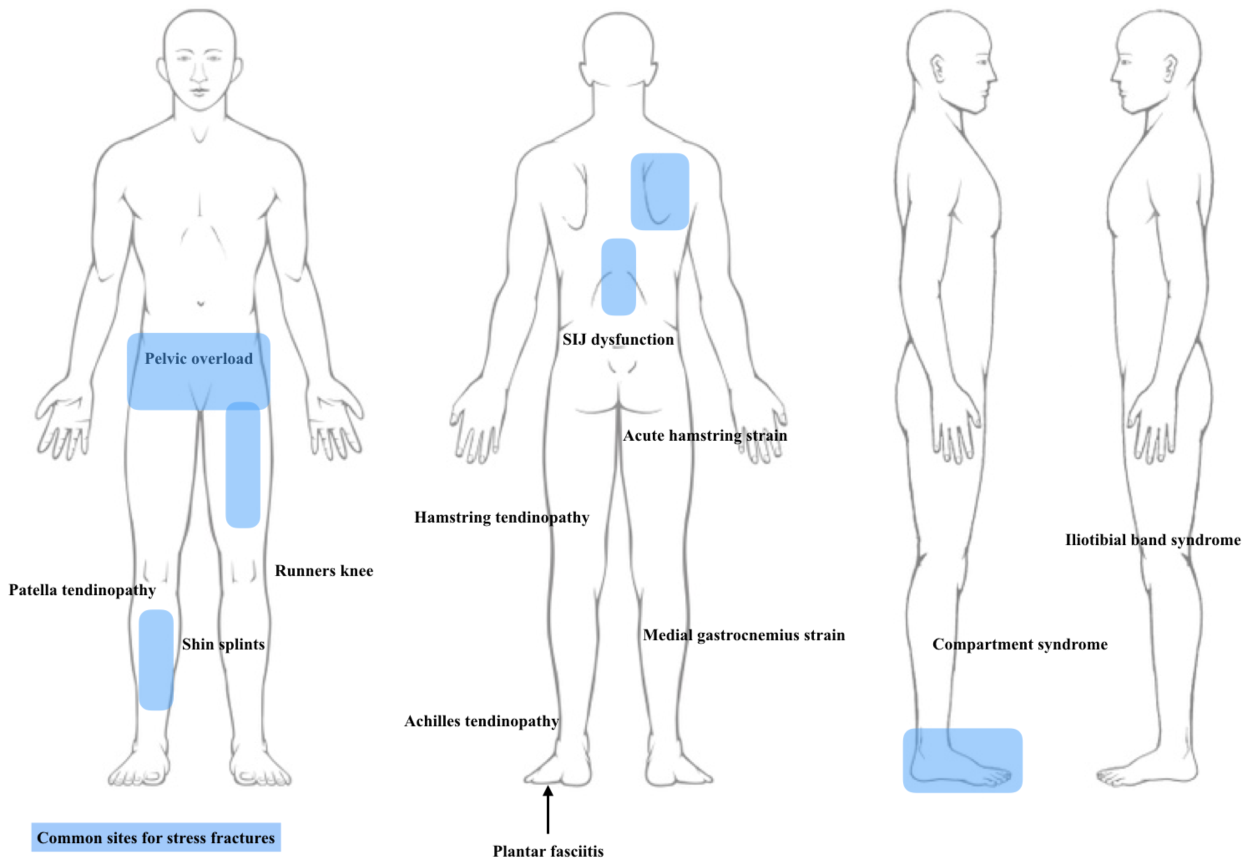


Figure 4.1: Common running-related overuse injuries and their location

ischemic[14] pain and possible lasting damage to the tissue in the compartment as well (Brukner and Khan, 2006; Diebal et al., 2011). *Iliotibial band syndrome* results from either friction generated between the *iliotibial band*[15] and the underlying lateral epicondyle[16] of the femur during knee flexion at foot strike, or compression of the fat layer between the iliotibial band and the lateral femoral epicondyle with the knee flexed below or close to 30º (Brukner and Khan, 2006; Louw and Deary, 2014). Overuse muscle strains of the calves (particularly the musculotendinous insertion of the *medial gastrocnemius*, as most force production during toe-off is directed alongside the medial part of the lower limb) (Pujalte and Silvis, 2014) and acute muscle strains of the hamstrings (related to sprinting) (Arnold and Moody, 2018) are associated with running.

The vast distribution of anatomical sites where injuries occur, and the connectedness of these injuries through the kinetic chain, attribute to the complex challenge to find interventions and long-lasting solutions for RROIs.

4.3 Risk and protective factors in running

Abundant literature is available to categorise risk and protective factors in running. One such framework is provided by Brukner and Khan (2006), who categorised the most general risk and/or protective factors for sporting activity: *internal elements* are neuromuscular, physiological, cardiovascular, genetics, biomechanics, psychological, previous injury and biometrics with *external elements* being the surface, weather, inciting event, equipment, opponents and

[14]deprived of blood flow
[15]the fibrous band on the side of the thigh
[16]bony protrusion on the outside of the knee

training load (see Figure 4.2).

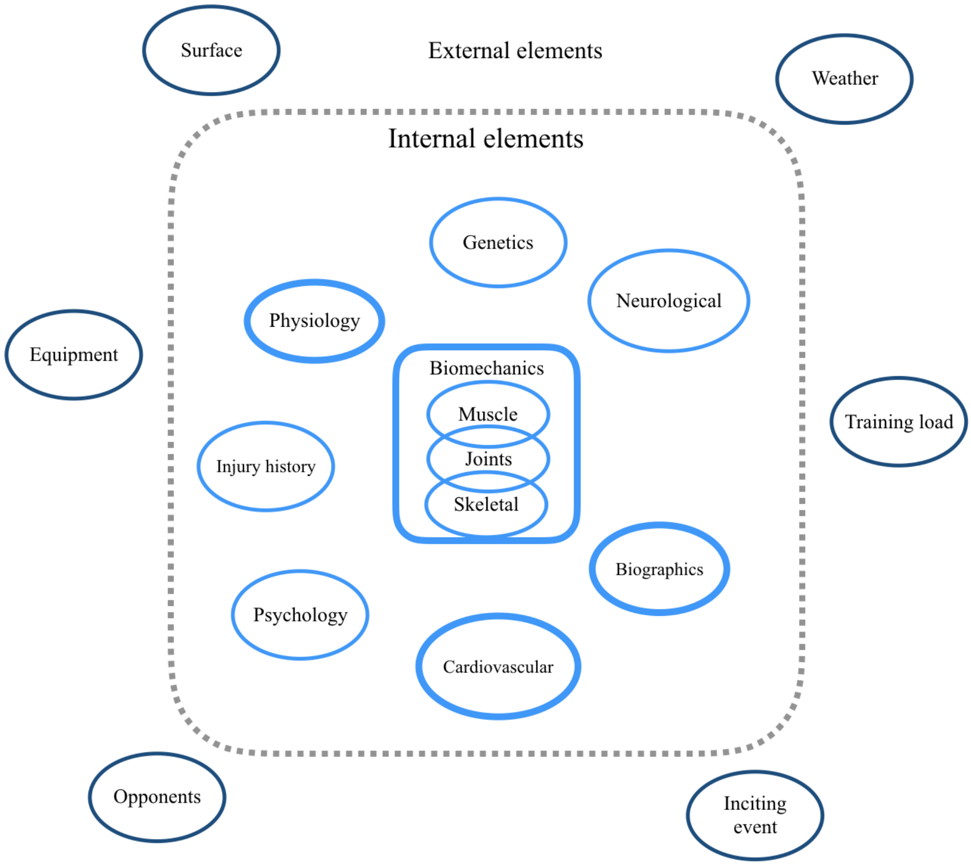


Figure 4.2: Generalised internal and external risk factors associated with injuries (Brukner and Khan, 2006).

Risk factors for running are classified in Larsen et al. (2016) as intrinsic, extrinsic and modifiable versus non-modifiable:

- *Intrinsic, modifiable:* personal factors included mechanical tissue properties of the MSS, psychological factors such as awareness, injury handling competency and training knowledge, training patterns and dynamic biomechanics. Joint alignments, tissue changes due to training and body composition may be modified through training or other mechanisms of conditioning.

- *Intrinsic, non-modifiable:* age, gender, anatomical properties of vital organs such as the size of the heart or leg lengths.

- *Extrinsic, modifiable:* training environment (surface hardness, choice of roads) and equipment (shoes, joint support braces and so forth.)

- *Extrinsic, non-modifiable:* the weather of the training environment (except if the athlete changes location), previous or concurrent injuries.

van der Worp et al. (2015) lists risk factors as follows:

- *Personal factors:* body mass index, height, weight, navicular drop, intratendinous blood flow, force distribution patterns and segmental joint alignment.

- *Running and training related:* experience, distance, surface, race participation and shoes.
- *Health and life factors:* History of previous injuries and the use of orthotics or inserts in shoes.

Molloy (2016) both concurs with the above but also adds fatigue from uncontrolled training bouts and transitioning in foot strike patterns. Nielsen et al. (2012) examined in a systematic review of the literature the relationship between training characteristics (volume, duration, frequency, and intensity) and RROIs. Relative risks, odds ratios, and hazard ratios for injuries generally increased for three studies with increasing weekly distance (volume of running ranged between less than five to more than 50 miles per week), whereas the injuries per 1000 hours of running recorded in one study decreased as volume increased. Odds ratios and relative risks were also higher for training frequencies of six or seven days per week when compared to five and/or less days per week. There were conflicting results with regards to incidence of RROI and intensity of training, with one study reporting higher risk of injury for paces faster than 8 $min/mile$, while others found no significant relationships between pace and likelihood of injury (Nielsen et al., 2012). Inadequate rest between training bouts and poor periodisation also predispose an athlete to higher risks for injury (Brukner and Khan, 2006). Recently, underlying chronic disease (of various organ systems, including the CVS, respiratory, neurological, heamatological and the gastrointestinal tract) and medication use (specifically, statins) were found to be novel risk factors associated with exercise-associated muscle cramping in distance runners (Schwellnus et al., 2018).

Hulme et al. (2017a) categorised risk- and protective factors for running into modifiable and non-modifiable categories, each with sub-categories in a systematic review of the available evidence. Modifiable sub-categories included stretching, warm-up and cool-down, surface and terrain, footwear, orthotics and insoles, diet and hormonal, body mass index, weight, distance, pace and intervals, frequency, duration, and psychological factors. Of these, only irregular menstruation or amenorrhea and not having used oral contraceptives were associated with higher risks for stress fractures in the female athlete. The non-modifiable sub-categories included age, height, sex, previous injury, experience, and biomedical factors. For this category, only a history of a previous injury was consistently associated with increased risk for a RROI.

Specific biomechanical and physiological risk factors for running include navicular drop in the foot, arch index of feet (indicating flat to highly arched feet), leg length discrepancies, knee and/or ankle varus and valgus, being overweight (van der Worp et al., 2015; Brukner and Khan, 2006), intratendinous blood flow (Via et al., 2016), over-striding (due to low cadence) (Heiderscheit et al., 2011), skeletal bone density (Astur et al., 2015), shortened and weak hamstrings, patellar maltracking due to incorrect motor unit firing of the quadriceps group (Dixit et al., 2007), pelvic instability, weak hip extensors, shortened hip flexors (Via et al., 2018), weak and shortened plantar flexors[17], inadequate aerobic fitness, poor nutrition (Maffetone and Laursen, 2016; Romani et al., 2002), weak intrinsic[18] foot muscles (Brukner and Khan, 2006), and degree of pronation and supination of the foot (Brukner and Khan, 2006; Nigg et al., 2015). Work in biomechanics to determine intrinsic and extrinsic risk factors in running are witnesses against each other (van Gent et al., 2007); evidence for the relationships between kinematic, flexibility and kinetic variables for good running economy and performance remain disputable (Moore et al., 2012; Trowell et al., 2019; Wen et al., 1997).

Tables 4.1 to 4.3 map the risk and protective factors to the classification framework from Brukner and Khan (2006) (seen in Figure 4.2).

[17] muslces that bend the foot downwards
[18] small muscles between the bones of the foot

Table 4.1: Internal risk for and protective factors against RROIs – biomechanics and cardiovascular

Category	**Specific risk factor**
Biomechanics[1]	Segmental joint alignments (varus or valgus of the ankles, knees and hips, spinal curvature; patella tracking) Height (manual entry) and leg lengths[2] Foot: arch index and navicular drop Force distribution patterns Cadence and stride length Vertical oscillation Foot strike pattern: heel-, mid-foot or front-foot landing Muscle strength and stability of: intrinsic foot, the pelvic and hip complex, the knee complex, the ankle complex, core stability Leg stiffness (may be approximated through adjustments in VO)
Cardiovascular[1]	VO_2-max Vascularity Heart function (or dysfunction) through HR and HRV Lactic acid threshold

[1] It is possible to extract some quantified values of variables from the overground tracking data from a RW.

[2] Adrian and Cooper (1995) excludes leg lengths as a risk factor *during running*, since there is no stance phase (when both feet are in contact with the ground). During walking, leg length discrepancy would cause a limp and mal-alignments elsewhere in the body, which are risk factors.

Table 4.2: Internal risk for and protective factors against RROIs – genetics, physiology, and history

Category	**Specific risk factor**
Genetics	Anatomical properties (example, size of heart and bone structure)
	Tissue mechanical properties
	Immune system
Physiology	Weight
	Intratendineous blood flow
	Metabolism (catabolism and anabolism) and energy balance
	Bone density
	Fatigue
	Ventilation capacity (lung function)
	Tissue repair and neo-tissue genesis (adaptive homeostasis)
	Quantity and quality of sleep
	Underlying chronic disease
Psychology	Experience (time the athlete has been running; total distances covered)[1]
	Fear of re-injury
	Adherence to coach (or practitioner) training advice (load management)[1]
	Awareness
	Injury handling competency (including patience during rehabilitation after injury)
	Motivational drive: internal or external
Biographics	Age and gender
Injury history	Scar tissue and biomechanical maladaptation

[1] Possible to be inferred from overground tracking data

Table 4.3: External risk and protective factors against RROIs

Category	**Specific factor**
Surface[1]	Hardness Surface profile: bends, straight, even, uneven ('lumpy'), obstacles, road camber Slopes (geography of the terrain)
Weather[2]	Temperature Rain Wind Humidity
Training load	Distance Number of steps At altitude, or elevation gained Frequency of training bouts and recovery time[3] Intensity Periodisation[3] Cross-training
Inciting event[4]	Falls (slippery surface, misstep on uneven terrain, bumping and shuffling in the pack)
Opponents	Tactics and size of the race field[2] (number of other participants that occupy the same space)
Equipment	Shoes and orthotics (inserts and/or braces) Compression wear Wearables
Diet	Nutritional intake, medication and substance abuse
Social media and popular literature	Online material available on *Youtube*, self-help guides that promote training strategies, social-sport platforms (example *Strava*)

[1] Possible to be inferred from overground tracking data through map-based GPS inspection
[2] Possible to be extracted from external data sources and linked with date from the RW tracking data
[3] Periodisation and recovery underscore the control over and management of the training schedule
[4] Possible interaction with underlying RROI

4.4 Domain specific solutions towards injury prevention and performance improvements[19]

Interventions to protect against injury and augment performance include both those with short and longer time horizons regarding the body's response and the efficacy of the intervention. Two types of interventions are considered in this section: physical resources as external aids and biomechanical interventions, both of which have been influenced by technology. Physical resources for running may be considered as minimal when compared to other individual sport such as cycling, yet many technological advances in running equipment have influenced the sport.

4.4.1 Technological advances in running equipment

Running shoes, as an influential variable of running outcomes for shod runners, and consumer wearables, as accessories, both contribute towards performance goals and injury protection. Recently, controversy evolved in the running community when sports equipment manufacturer Nike released the Vaporfly series running shoes, featuring advanced material technology and innovative architectural construction of shoes. Claims have been made that the shoe improves running economy by 4%, and may increase a runner's speed by 3.4% (Burns and Tam, 2019), while others reported a 4.2% +- 1.2% improvement in running economy when compared with another long distance running shoe from competitor Adidas (Barnes and Kilding, 2019). Both men's and women's records in the long distance races have recently been broken by considerable margins, with the athletes running in the Vaporfly (Guinness et al., 2020).

Sports journalism reported how the shoe provides runners with an unfair advantage, in some instances likening the shoe's enhanced energy return as 'mechanical doping', whereby increased performance are attributed to the shoe's action and not the inherent capabilities and training efforts of the athlete. There were expectations that the shoes may be banned in road running due to the unfair advantage and shifts in the playing fields (Philips, 2020; Kilgore, 2020; WSJ, 2020; CBSN, 2019). Instead, shoe regulations have been suggested to allow space for manufacturers to be innovative and advance the sport, yet not provide any unfair advantage (Burns and Tam, 2019).

Consumer wearables have made visible to runners what was for most part only available through laboratory tests or tedious manual observation. These wearables (fitness trackers, running watches and smart watches) have become popular amongst runners to self-monitor some biomechanical and physiological variables as a means to improve on running gait (for example, cadence and VO) (Passfield and Hopker, 2016; Vermeulen, 2018) and to gauge recovery through HR and HRV monitoring (Reali et al., 2019). Complimentary social media platforms enhance data analysis, enable performance sharing, encouragement, competition and digital coaching.

However, the researcher argues that, though well intended, wearables and fitness related social media may serve as either protective or risk factors: protective since runners gain information to complex concepts to enable better understanding in athletic health and injury development. Nonetheless, wearables and platforms may contribute to running injuries: often the views (sometimes personal and not from a professional background) expressed on social media promote single, polarised strategies towards healthier running, injury management or recovery, thereby neglecting the interactions between the many units that make up the ALS. Data from consumer wearables are not fully reliable with low signal-to-noise ratios (Karkazis and Fishman, 2017), providing bias information to the athlete and subsequent wrong training

[19]parts of this section were published in *Theoretical Issues in Ergonomics Science*, July 2020

decisions may be made.

4.4.2 Gait retraining: running is a skilled activity

Although running comes naturally to humans, an ideal running gait is an acquired skill which must be learnt through interaction within the neuromuscular pathways, the MSS (Barton, 2018) and the CVS to produce good performance and minimise risks of RROIs (Maffetone and Laursen, 2016). Gait retraining strategies are described as "the implementation of any cue or strategy to alter an individual's running technique" (Barton (2018):79).

Running biomechanics on which gait retraining stratgeies are based, are summarised from Barton et al. (2016) and investigated in more detail in other work:

- Foot strike pattern (Larson, 2014; Shih et al., 2018; Mercer and Horsch, 2015)
- Stride frequency (Schubart et al., 2014; Adams et al., 2018)
- Stride length (Heiderscheit et al., 2011; McArdle et al., 2010)
- Stride width (cross-over gait) (Brindle et al., 2014; Meardon and Derrick, 2014)
- Vertical oscillation (Adams et al., 2018; van der Worp et al., 2016)
- Trunk posture (head alignment with the trunk) (Shih et al., 2018; Teng and Powers, 2014),
- Hip drive and pelvic motion (Dingenen et al., 2019; Bramah et al., 2018)
- Knee drive
- Knee-toe alignment
- Leg stiffness (Girard et al., 2013; Shih et al., 2019)
- Barefoot and shod running (Larson, 2014; Sinclair, 2014)

Barton (2018) accentuate that clinical evidence for the effectiveness of exercise therapy as adjunctive to gait retraining in the long term is lacking, citing several reasons, one being that the time horizons of interventions are too short to truly appreciate the long term effect. This statement reflects the ST concept of dynamic complexity: when the same action elicits different effects in the short and long term (Senge, 1990). That is, there is a strong time component, i.e. delays, underlying exercise therapy and gait retraining: cause and effect is distanced in time, taking away visibility of efficacy and possibly curtailing motivation to continue with the intervention in practice. Well-trained athletes have through experience learnt the optimal combination of stride frequency and stride length which they are accustomed to and which yields the most economical performance (McArdle et al., 2010; Moore et al., 2012).

Of the 46 studies included in Barton et al. (2016), the large majority of studies focused on stride frequency and foot strike patterns (19 and 18 respectively) and only one study focussed on a combined approach of the strategies above. Although gait retraining is an attempt to change the behaviour of the ALS from the interaction between structural units of muscles, joints and limbs, this distribution of work in gait retraining shows that research in the field is perhaps still following a reductionist approach by excluding the interactions *between* the strategies and their feedback to other units of the MSS. For instance, increasing stride frequency without an optimal trunk posture may simply have the runner pounding the leg into the ground upon landing, accompanied by a pelvic drop, instead of capitalising on momentum from a forward lean and the elastic recoil offered by leg stiffness and a softer landing.

Transitioning to barefoot running (or minimalist shoes) from traditional cushioned running shoes have recently received the attention of sport science and the general running community. Minimalist shoes have been questioned in their ability to mimic or simulate barefoot running, whilst also linked to increases in bone marrow oedema of the foot bones when transitioning to minimalist footwear (Tam et al., 2017). Foot strike patterns and loading rates are mostly studied. Most shod runners employ a rear-foot strike pattern (Larson, 2014; Hashish et al., 2016) while habitual barefoot runners favour a front-foot strike pattern (Hashish et al., 2016). Larson (2014) found 59.2% of barefoot runners to forefoot strike in a recreational race on an asphalt surface. The benefits of barefoot running is purported to be the shift in foot strike pattern, accompanied by decreased loading rates which lowers injury risk exposure. However, transitioning from a rear-foot strike to a mid- or forefoot strike shifts the mechanical demand from the knee to the ankle, exposing the Achilles tendon (and the calf musculature) to unaccustomed loading rates. This shift increases the risk of injury to the Achilles and calf musculature (Sinclair, 2014).

Hashish et al. (2016) studied the natural responses of novice barefoot runners who transitioned from traditional running shoes. The group of 22 runners responded heterogeneously and was divided into those who maintained a rear-foot strike (8), some transitioned to mid-foot strike (9) and some to a forefoot strike (5). Subsequently, those who maintain a rear-foot strike experienced increases in loading rates (108%) when transitioning from shod to barefoot running, while the forefoot strikers showed a 41% decrease in loading rate. Barefoot running reduced energy absorption at the knee for all groups, although energy absorption at the ankle increased by 140% for front-foot strikers. The increase in loading rate for those who maintained the rear-foot strike predisposes these runners to injury, whilst the sudden shift in loading rate to the ankle for front-foot strikers also increases risk for injury in the unprepared Achilles tendon and calf musculature (Hashish et al., 2016). To reap the benefits of barefoot running, a gradual guided process (specifically the change to a more forefoot strike pattern) is needed and runners must become accustomed to the new loading rate of the ankle, allowing adequate time for the muscles to adapt to the demand. Additional strengthening is advised, along with gradual increases in barefoot running volumes (McCarthy and Fleming, 2015).

4.5 The higher cadence strategy: the double-edged sword in gait-retraining

Runners alter their running form for mainly two reasons: to adjust speed (Lieberman et al., 2015) or to prevent injuries from repetitive high loads (Barton et al., 2016). Running form is mostly defined by two biomechanical characteristics: stride length and stride frequency (Heiderscheit et al., 2011; McArdle et al., 2010). Here, the runner's stride is the distance covered between footfalls of the same foot. Stride frequency, or cadence, is the number of strides taken per unit time. It follows that a runner's pace (as a reciprocal of speed) is a function of their stride and the frequency thereof in Equation 4.1:

$$Pace = \frac{1}{\text{stride} \times \text{cadence}} \tag{4.1}$$

Pace (time per distance) may therefore be increased (become faster) through a higher cadence, a longer stride or some combination strategy.

Both stride and cadence is achieved in the runner's unique gait pattern, which is self-optimised based on energy production for locomotion (Moore et al., 2012; Moore, 2016; McArdle et al., 2010). Modulation of stride length and VO away from the self-selected gait may negate running economy (Moore et al., 2012), increasing the risk of injury due to fatigue. How-

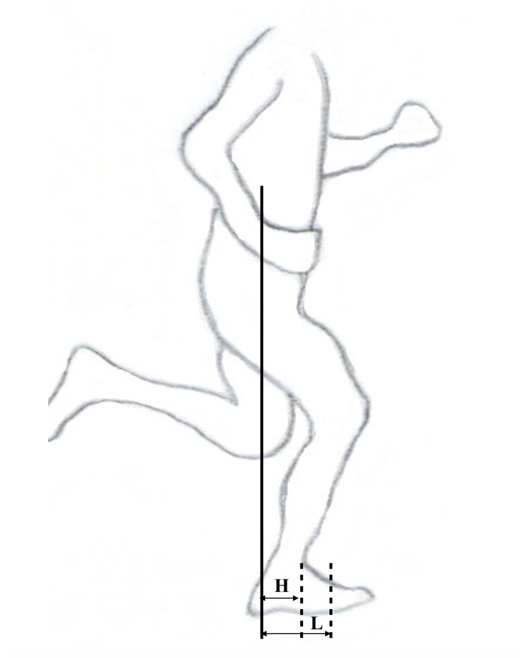


Figure 4.3: Torque arms

ever, the runner's self-optimised gait is based on energy conservation (McArdle et al., 2010; Snyder and Farley, 2011; Lieberman et al., 2015), not accounting for force distribution and load management of the lower limbs. Figure 4.3 shows the lengths of the moment arms of the ground reaction force on the hip when the heel is underneath or close to the hip upon footfall (arm H) and further away (arm L), landing with the foot in front of the hip.

Greater torque is produced up the kinetic chain by the longer arm L, than H, resulting in more negative work required in the hips and knees when the foot makes contact in front of the hip's plumb line (Levangie, 2005). Increasing the cadence will place the foot closer to the hip's vertical line upon landing (moving from L to H) and reduce the negative workload in the hips and knees (Heiderscheit et al., 2011; Lieberman et al., 2015). The foot strike pattern subsequently changes, transitioning from heel strike towards the mid-foot or front-foot landing. The breaking force incurred by the foot during ground contact is reduced when cadence is increased and a transition is made to mid- or forefoot strike, with a more vertical tibia (Lieberman et al., 2015).

This change in foot strike pattern is the double-edged sword when running gait is modified through the higher cadence strategy (Molloy, 2016). Elastic, eccentric loading of the ankle plantar flexors (the calve muscles) increases (Barton, 2018) because the fulcrum of the GRF lever has changed to the ball of the foot (Levangie, 2005), so that the lower limb functions more spring-like to propel the body forward. The load (also the negative work) therefore moves away from the knees and hips, but is transferred to the ankle and foot (Molloy, 2016; Barton, 2018) while the hip flexor moment increases during the swing phase (Lieberman et al., 2015).

The MSS and the CVS must now be considered within the new force distribution pattern. Through exercise therapy (focussed on strengthening and flexibility training), the mechanical properties of the MSS in the lower limbs should adapt to deal with the higher dynamic loading at the involved joints (Barton et al., 2016; Brukner and Khan, 2006); otherwise face overloading injuries of the soft tissue in the MSS such as tendinopathy of the plantar flexors (Molloy, 2016) or medial tibial stress syndrome, which may progress into a tibial stress fracture (McClure and Oh, 2019). Conversely, a tibial stress fracture is one of the injuries that a higher cadence should protect against (Adams et al., 2018).

From § 3.1.1 *The stress-strain curve*, the stress-strain curve (in Figure 3.3) of the *plantar* *flexors* must shift upward, undergoing an increase in its elasticity (steeper slope of the linear portion) and handle higher stress at the same strain (deformation) in the plastic region. That is, an increase in leg stiffness is required (Farley and González, 1996; Girard et al., 2013).

Furthermore, the angular velocity of the lower limbs' levers around the hip and knees must be increased during swing (Heiderscheit et al., 2011; Lieberman et al., 2015), requiring greater pelvic and knee stability upon landing to limit VO (Wang et al., 2012). The CVS must respond with augmented vascularity, through higher circulatory distribution and neo-genesis of blood vessels at the involved muscles to supply the needed fuel and oxygen from shifts in demand (McArdle et al., 2010).

The transitioning to a higher cadence is promoted in the literature (Heiderscheit et al., 2011; Schubart et al., 2014; Adams et al., 2018) as protective against repetitive load related injuries due to the decrease in negative work at the hips and knees. However, altering the running form of an athlete has a metabolic cost attached (Snyder and Farley, 2011; McArdle et al., 2010). McArdle et al. (2010) show that athletes have an optimal stride length to coincide with minimum oxygen consumption. The oxygen-consumption-stride-length function takes on a u-curve shape: at a constant pace, oxygen consumption is minimised at an optimal stride length, and increases on either side. That is, whether the stride is lengthened (stride frequency decrease) or shortened (stride frequency increased), oxygen consumption increases away from the athlete's optimal point. Similarly, Lieberman et al. (2015) showed a u-shaped curve for energetic costs and cadence, with the net energetic costs being minimised at the optimal cadence.

The adaptations in the mechanical properties of the MSS and a modified running gait should be the outcome from *tailored* physical training (Napier et al., 2017; Barton, 2018) to avoid new, unintended injuries during and after such a transition. It will take time for the structures in the ALS to adapt to the new energy demand patterns that a higher cadence asks from the MSS and the CVS. Initially, performance may be negated by the increase in metabolic costs, and an athlete may struggle to maintain the higher cadence for a sufficient time so the altered gait becomes profitable (Heiderscheit et al., 2011). Disregarding the athlete's body unique response (from both the MSS and the CVS) to altered loading may become the cause of another injury.

The physiological intensity of running changes with increases in cadence, and results in the onset of fatigue to occur earlier(Adams et al., 2018). An athlete must structurally adapt to the intensity before taking on higher volumes of training. Opting for higher training volumes in the absence of strengthened structures to deal with the new level of intensity, predisposes the athlete to overuse injuries at a different site (Barton et al., 2016; Barton, 2018). Adjusting cadence as a means to achieve lower loading rates in order to protect against injury, remains subject to conditioning and a gradual transition.

4.6 The overtraining syndrome

Maffetone and Laursen (2016) regard physical injuries (some neuromuscular and biomechanical dysfunction), biochemical injuries (endocrine and immune system dysfunction) and mental-emotional injuries (depression) as non-normal outcomes from participation in endurance sport, yet the frequency rate of such injuries among athletes remains high. Effective training is the combination of physical overload to induce desired physiological modulations and recovery to allow for these modulations. An unhealthy situation develops when the carefully balanced scale between overloading and recovery is disturbed and tips in favour of overloading while neglecting recovery (Kreher and Schwartz, 2012). The OTS is defined by Kreher and Schwartz (2012) as the maladaptive response to immoderate exercise coupled with inadequate recovery, culminating in perturbations of bodily systems (immunologic, neurologic and endocrinologic). Meeusen et al. (2013) describes OTS as the accumulation of training induced stress with insufficient recuperation, resulting in long-term performance decrements; restoration of physical capacity to perform may take months.

The OTS is characterised by a collection of symptoms, but not all symptoms are necessarily present at the same time. Common symptoms in aerobic sport, such as distance running, are mostly parasympathetic in origin such as fatigue, depression, bradycardia, loss of motivation, and further include heavy and sore muscles, anxiety and lack of concentration (Kreher and Schwartz, 2012). The aetiology for OTS is subject to a multiple of hypotheses (Kreher and Schwartz, 2012) and varies amongst ability (recreational to elite), age, gender, sport type (aerobic or anaerobic) and levels of training history (Maffetone and Laursen, 2016).

Athletes, coaches and practitioners must remain cognisant of their physical health, and not just their fitness levels. Ignoring these symptoms or ascribing them as 'normal' after intense training will eventually lead to the exact opposite of the desired outcome from training: a chronically fatigued athlete, continued underperformance and at greater risk for physical injuries (Maffetone and Laursen, 2016).

Treatment for OTS is rest (although relative rest is promoted). Pharmacological treatment includes serotonin reuptake inhibitors (Kreher and Schwartz, 2012). Early identification of OTS largely relies on monitoring performance and training load in conjunction with stress indicators, such as the Profile of Mood States (Kreher and Schwartz, 2012). Other monitoring methods include training diaries, physiological screening, retrospective questionnaires, psychological screening, and the rate of perceived exertion (Meeusen et al., 2013). Prevention of OTS includes adequate balance of training loads, individualised training intensity, sleep, rest periods (more than six hours between training bouts), abstinence from training following infection and/or heat stress, sufficient hydration and nutrition, good periodisation, development of mental resilience, avoidance of excessive monotony in the training regime, and management of environmental and life (interpersonal) stressors (Kreher and Schwartz, 2012; Meeusen et al., 2013). Despite large investments made into the research in overtraining, the OTS remains a difficult phenomenon to monitor and prevent.

4.7 Environmental stressors in running

Environmental factors originating from the atmosphere (weather and oxygen content at altitude) may not result in RROIs *per se*, nonetheless through interaction with the MSS and the CVS they contribute to RROIs. The geography of the terrain in terms of surface gradients, profile and the type of surface may perhaps more directly contribute to RROIs.

4.7.1 Gradient running, surface stiffness and surface profile

From a physics and mechanical perspective, three external structural elements, the gradient of the road, surface stiffness, and the surface profile (even, uneven or cambered) influence the biomechanics of the MSS by changing the length and orientation of the limb levers in relation to each other. Put otherwise, the joint alignments are altered with subsequent changes in muscle length and strain during action.

Vermeulen (2018) provide a short summary of the effects of gradient running on the dynamic elements of the WoD, specifically cadence. There is not yet consensus in literature as to how runners change their cadence (increasing or decreasing) when running uphill or downhill in comparison with level running. Some studies discussed in Vermeulen (2018) claimed increases in cadence (one study reported an increase of 0.04 strides.min^{-1} while others showed 4% increases in cadence when the slope changed from 0% to 7%) and others reported no changes. That there is no consensus about the runner's cadence during gradient running is another witness to the complexity of the ALS's unique biomechanical footprint as the runner move overground.

Runners adjust their leg stiffness (see § 5.1.4 *Human leg stiffness, k* for a formal definition of leg stiffness) in reaction to the stiffness of the surface encountered, thereby maintaining similar biomechanics between different surface types (Ferris and Farley, 1997; Wang et al., 2012). Muscular actions must therefore adapt to still recycle the elastic energy during the length-shorten cycle of leg turn-over, although the elastic return of the surface might be diminished in compliant surfaces (for example grass, trail, gravel or sand) or have increased on stiffer surfaces (asphalt or concrete) (Feehery, 1986). Ferris and Farley (1997) reports leg spring stiffness adjustments of up to 3.6-fold to accommodate less stiff surfaces. On surfaces with stiffness 35 000 kN/m, leg stiffness adjusted to 18.9 kN/m at a hopping frequency of 2.2 Hz. A more compliant surface (26.1 kN/m) was associated with higher leg stiffness (53.3 kN/m) at a hopping frequency of 2 Hz. Leg spring displacements ranged from 0.138 m for the stiff surface to 0.043 m for the least stiff surface. The human runner prefers to increase leg stiffness on the compliant surfaces and decrease stiffness on harder surfaces (Ferris and Farley, 1997).

Different plantar loads were recorded for runners running overground on natural grass, synthetic rubber surface (i.e. an artificial track) and concrete in Wang et al. (2012). The lowest maximum plantar pressure was found for grass, followed by the synthetic rubber surface with the highest maximum pressure found for running on concrete. The differences in plantar pressure indicate kinematic changes through active adaptations in the MSS to adapt to the different energy return from the surface in order to uphold similar impact (Wang et al., 2012). Tessutti et al. (2012) had similar results, recording peak pressures 9.3% to 16.6% lower on grass than on rubber, concrete and asphalt in the rearfoot and 4.7% to 12.3% reduction in the forefoot.

The kinematic changes result in (perhaps small) alterations of the moment arm of the ground reactions forces through the joints during stance (Wang et al., 2012). Running on concrete, runners had increased flexion in the ankles, knees and hips at heel-strike. Increases in flexion augments the muscle forces required to maintain locomotion. Long distances covered over concrete with the high plantar pressure may lead to repetitive damage on structures, leading to a RROI (Wang et al., 2012).

Wang et al. (2012) concludes that running on natural grass may be protective against RROIs, due to the lower plantar pressure. Tessutti et al. (2012) supports this statement, adding that running on grass (under a controlled amount of intensity) may reduce the total stress on the MSS through the attenuated pressure on the feet when compared to hard and rigid surfaces.

Voloshina and Ferris (2015) reported an increase of 20% in leg stiffness during running on uneven terrain when compared to even terrain in their study involving 12 human subjects. Unfried et al. (2013) found differences in muscle activities in the lower extremities during stance phase on a cambered surface. The muscles *tibialis anterior, lateral gastrocnemius*, and *vastus medialis oblique* showed higher activity during stance on the gutter side of the road than on the crown size. The disparities in muscle activities are assigned to compensatory response of the lower extremity levers to the cambered condition. Long-term compensations coupled with the repetitive impact of running on the altered lever orientation of the lower extremities will cause additive damage to the structures of the MSS, leading to a RROI (Larsen et al., 2016; Herring and Nilson, 1987; van der Worp et al., 2015).

Runners interact with the surface they encounter and adjust their biomechanics according to the compliance and profile of the surface. Such adaptations may be to the benefit of some structures, but come at a cost to others.

4.7.2 Heat illness or injury

Mentioned earlier in § 3.1.3 *The cardiovascular system* and § 3.1.4 *Thermoregulation*, aerobic fitness is protective against heat illness with enhanced thermoregulation in response to sudden heat stress and allows for easier acclimatisation to develop tolerance. Nonetheless, aerobic fitness alone will not protect against overheating; complexity may arise in superior fit athletes (Laitano et al., 2019).

Heat stress may induce three types of illnesses of varying severity and fatality. Heat cramps occur when selective minerals are not available to muscles for normal contraction-relaxation cycles, resulting in sustained contraction or spasms. Improved hydration and nutrition will relieve and reduce spasms. Heat exhaustion results from inefficient circulatory adjustments to heat stress. Blood pools in the dilated vessels in the periphery, subsequently blood pressure decreases and CO is not effectively distributed. Acute treatment includes moving to a cooler environment, cessation of activity, and intravenous fluid replacement if needed (McArdle et al., 2010).

In heat stroke, the total heat load (metabolic and/or environmental) has exceeded the heat dissipating mechanisms and thermoregulation fails, presenting a life-threatening emergency (Nichols, 2014). Exertional heat stroke is the result of complex interactions between internal metabolic heat produced during physical activity and external agents (mostly a hot, humid environment) that limits heat dissipation; in classic heat stroke, it is the environmental heat that overloads the heat dissipating mechanisms (McArdle et al., 2010; Laitano et al., 2019). In either case, heat is accumulated and the core temperature (T_r) rises dangerously high.

Literature is inconclusive with regards to the boundary values of T_r, to distinguish between heat stroke and exertional heat stroke, but generally $T_r > 40.5^o$ C for classic heat stroke and $T_r > 41.5^o$ C for exertional heat stroke (McArdle et al., 2010; Nichols, 2014; Laitano et al., 2019). Cascading systemic tissue damage ensues with injuries to skeletal muscles and organ damage from the failure of the CVS and the nervous system. Heat stroke leads to death when left untreated or cooling is delayed (McArdle et al., 2010; Nichols, 2014), with exertional heat stroke being the third leading contributor to death amongst athletes (elite or recreational) (Laitano et al., 2019).

The runner interacts with the atmospheric conditions in order to maintain both homeostasis and locomotion. In some instances, such as under severe heat stress, the body maladapts or is overwhelmed. Subsequently, damage (in some severe instances, fatal) to structures follows under these conditions.

4.8 Conclusion

This chapter described the risk and protective factors for running, musculoskeletal RROIs, the OTS and environmental stressors for running. Environmental stressors either can contribute to fitness or negate it, depending on how it is leveraged by the athlete. The volume, intensity and recovery periods between training bouts are to be carefully considered in a well-balanced training programme, as to produce a healthy athlete fit for performing the task at hand. Imbalances in the training programme where recovery is neglected negate any fitness an athlete has accumulated.

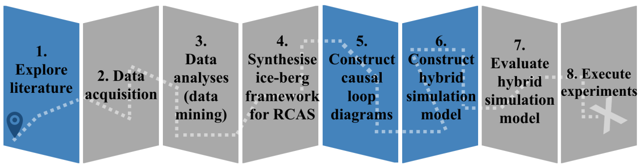
Incidence of RROIs is persistent despite numerous attempts to quantify risk and/or protective factors as well as evaluating potential effective interventions. The chapters to follow are dedicated to practically illustrate a novel approach to the problem: principle tools from ST and dynamic modelling.

Chapter 5

The laws of physics applied to running

Contents

This chapter formulates parts of tasks 1, 5, and 6 from the research methodology.



The scientific and mathematical derivation of the methods applied in tasks 5 and 6, are unpacked in this chapter. In preparation for the qualitative and quantitative models, the spring-mass system, how energy is recycled, impulse generation, and entropy of a living system are analysed in this chapter. The runner is modelled as a mechanical spring-mass system that does work to propel the runner into the air and overground. The micro-damage-to-adaptation route will be modelled within the context of entropy of a living system, whereby disorder first increase, followed by adaptation. Uncontrolled, cumulative disorder leads to injury; however, when disorder is removed by introducing negentropy, the runner adapts and becomes more fit. Part of entropy, another concept is introduced at the intersection of tracking data from wearables and adaptation: the arrow of time.

5.1 The runner as a spring-mass system

The behaviour of the muscles, tendons and ligaments of the lower limbs during running is much similar to the mechanical behaviour of a linear spring-mass system (Farley and González, 1996; Ferris and Farley, 1997; Morin, 2018; Girard et al., 2013; Moore, 2016). The human body is complex and can be anatomically divided into several spring-mass components (Clark et al., 2017), which work together during locomotion so that the overall MSS behave as a single spring-mass system. In this system, the total body mass acts as a point-mass attached to springs, represented by the legs (Ferris and Farley, 1997; Girard et al., 2013; Kulmala et al., 2018).

As mechanical applications, springs are used to absorb forces, and store the energy as elastic potential energy, which is released when the applied force is removed (Halliday et al., 2011).

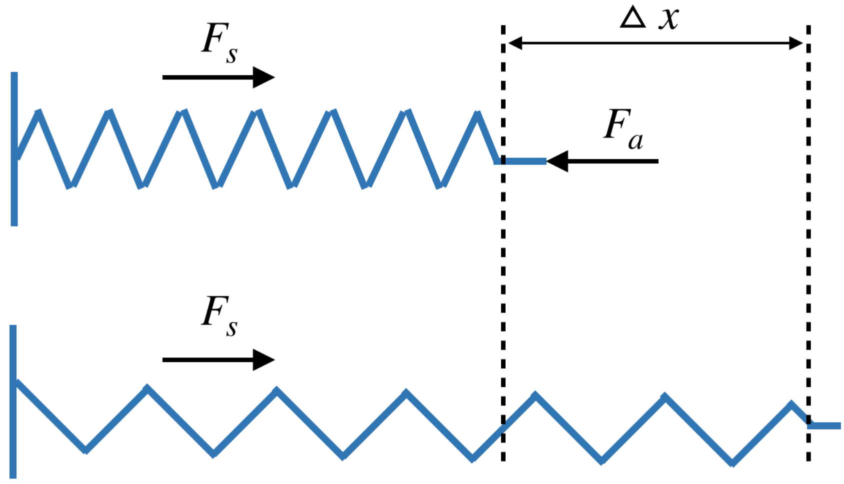


Figure 5.1: Compressed spring

5.1.1 Work and elastic potential energy

The elastic potential energy, E_{pt} *"is the energy associated with state of compression or extension of an elastic object"* (Halliday et al., 2011, p.167), such as the spring. Halliday et al. (2011) explains how a spring works (see Figure 5.1). A force is applied (F_a) to the spring, to either compress the spring or elongate the spring. There exists a reaction (or restoring) force, F_s, in the opposite direction of the applied force (F_a) to return the spring to its neutral length, i.e. the spring recoils back to its original length. From Hooke's law, the force F_s of a spring is a function of the spring's elastic constant (the load constant, or the stiffness coefficient) k and the displacement $\triangle x$ of the spring as it is compressed or elongated:

$$F_s = -k \triangle x \tag{5.1}$$

The spring force (F_s) is a variable force as it proportional to the spring constant k. Work (W_s) done by the spring is the integral of the force applied over the change in length x from the neutral position of the spring:

$$\begin{aligned} W_s &= \int_{x_i}^{x_f} F_s \, dx \\ &= \int_{x_i}^{x_f} -kx \, dx \\ &= -\frac{1}{2}k(x_f^2 - x_i^2) \end{aligned} \tag{5.2}$$

Negative work will be done by the spring force when the spring is stretched out ($\triangle x > 0$; $x_f > x_i$), that is $W_s < 0$. Positive work will be done by the spring force when the spring is compressed ($\triangle x < 0$; $x_f < x_i$). Elastic energy can be stored in the compressed or elongated spring. Upon release, when F_a is removed, F_s remains and there is elastic recoil back to the spring's neutral length. The elastic potential energy is released to do work and is transformed into kinetic energy (Halliday et al., 2011).

5.1.2 Modelling the spring-mass system with simple harmonic motion

The repetitive up-and-down movement of the CoM of a runner represents a waveform function over time (Clark et al., 2017), and can be modelled as SHM. The analysis for SHM is taken from Halliday et al. (2011). An object that is moving back and forth (or up and down) around an axis is an oscillating object with two important properties: frequency (f, number of oscillations completed per second) and the period, T_c, which is the time taken to complete one oscillation (or cycle). Frequency is expressed in hertz (Hz):

$$f = \frac{\text{number of oscilations}}{\text{total time in seconds}} = 1\ s^{-1} = 1\ Hz \tag{5.3}$$

The period (T_c) is the time taken to complete one cycle and is the inverse of frequency:

$$T_c = \frac{1}{f} \text{ seconds} \tag{5.4}$$

The displacement, velocity and acceleration of periodic motion can be modelled as SHM, where the motion is a sinusoidal function of time. The displacement x is a function of time in a cosine waveform:

$$x(t) = x_m \cos(\omega t + \theta) \tag{5.5}$$

where x_m is the amplitude, ω is the angular frequency and θ is the phase angle. The amplitude is the maximum displacement from the central axis that an oscillating object travels. The angular frequency ω can be understood in terms of the displacement $x(t)$, where the cosine function returns to its starting position in one period T_c. The cosine function first repeat starts when the function has rotated through 2π. Simplifying the analysis, let $\theta = 0$ in Equation 5.5 and rewrite it as:

$$x_m \cos(\omega t) = x_m \cos(\omega(t + T_c)) \tag{5.6}$$

Then, at time $t + T_c$, from Equation 5.6 the relationship between angular frequency and the period is derived as follows:

$$\begin{aligned} \omega(t + T_c) &= \omega t + 2\pi \\ \omega T_c &= 2\pi \\ \omega &= \frac{2\pi}{T_c} = 2\pi f \end{aligned} \tag{5.7}$$

Velocity and acceleration can now be derived from displacement $x(t)$. Velocity is the first derivative of displacement:

$$v(t) = x'(t) = -x_m \omega \sin(\omega t + \theta) \tag{5.8}$$

Acceleration is the first derivative of velocity (or second derivative displacement):

$$a(t) = v'(t) = -x_m \omega^2 \cos(\omega t + \theta) \tag{5.9}$$

From 5.5 and 5.9, acceleration may be simplified to:

$$a(t) = -\omega^2 x(t) \tag{5.10}$$

The force law for SHM is derived from Equation 5.10 and Newton's second law:

$$F = ma = -m\omega^2 x \tag{5.11}$$

The spring-mass system acts as a linear oscillator as the mass is displaced back and forth from its equilibrium position and can be modelled as SHM. Equation 5.11 is reminiscence of Hooke's that describes the restoring force of the spring-mass system: $F = -kx$. The spring constant k is replaced with $m\omega^2$ from Equation 5.11, then:

$$k = m\omega^2 \tag{5.12}$$

It follows that the angular frequency ω in SHM relates to the spring constant k and the mass m from Equation 5.12, which can be rewritten as:

$$\omega = \sqrt{\frac{k}{m}} \tag{5.13}$$

The period for the linear oscillator is written as a combination from 5.13 and 5.7:

$$T_c = 2\pi\sqrt{\frac{m}{k}} \tag{5.14}$$

5.1.3 Impulse: a measure of a force's magnitude and duration

Impulse and the vertical force-time waveform patterns are studied to determine changes in running mechanics, running economy, limb loading rates, stress in tissues, and the body's state of motion (Girard et al., 2013; Lieberman et al., 2015; Clark et al., 2017). Impulse is a measure of the magnitude and application time of an impact force during a collision between two or more objects (Halliday et al., 2011). Newton's second law can be re-stated to say that the momentum p of a body cannot change unless there is a net external force acting on it to change its momentum. During collisions, when a time variable force F is applied suddenly and for a short period of time dt, the body's momentum undergoes a change, dp. (Halliday et al., 2011). Newton's second law can be rewritten as:

$$\begin{aligned} F(t) &= \frac{dp}{dt} \\ dp &= F(t)dt \end{aligned} \tag{5.15}$$

Integrating the right side, the force-time function $F(t)$, gives the impulse in N-s, the measure of the magnitude of the force and the duration of its application (Halliday et al., 2011):

$$I = \int_{t_i}^{t_f} F(t)\ dt \tag{5.16}$$

The linear-momentum-impulse theorem states that the net change in momentum is equal to the impulse on the object:

$$\begin{aligned} I &= \triangle\ p \\ &= m \triangle v \end{aligned} \tag{5.17}$$

During running, there is contact (or a vertical collision) with the ground when the foot strikes the ground, through stance phase and ends when the toe is lifted. The GRFs varies with time during the stance phase. During this time, the body has undergone a change in momentum. It decelerates from its highest aerial CoM position to its lowest position during ground contact, at the peak vGRF and then undergoes acceleration again (propulsion) to enter the flight stage (see Figure 5.3). The body is encountering cyclical impulse loading from these vertical collisions as it moves over the ground, alternating between being airborne and in contact with the ground (Kulmala et al., 2018; Clark et al., 2017).

5.1.4 Human leg stiffness, k

Human leg stiffness is derived in terms of Hooke's law (Butler et al., 2003), also referred to earlier in § 3.1.1 *The stress-strain curve* to model the elastic deformation of loaded muscles and tendons.

Definitions of human leg stiffness

Stiffness of the entire MSS during locomotion is described in Moore (2016) as the ratio of deformation (through vertical displacement of the CoM, $\triangle\ y$) to the vGRF acting on the body upon footfall in Equation 5.18:

$$k_l = \frac{vGRF}{\triangle\ y} \tag{5.18}$$

Then the MSS's restoring force (F_s, from Equation 5.1) is generated by the elastic deformation of muscles and tendons, in reaction to the applied $vGRF$. Rearranging Equation 5.18 in terms of the force F_s, a familiar expression emerges:

$$F_s = k_l \triangle\ y \tag{5.19}$$

Butler et al. (2003) distinguishes three categories of how stiffness may be calculated: vertical-, leg- and torsional stiffness. Vertical stiffness (k_v) is calculated using three methods, when the leg is orientated vertically to the ground. First, the maximum force during stance and the displacement of the CoM is used:

$$k_v = \frac{F_{max}}{\triangle\ y} \tag{5.20}$$

where $\triangle\ y$ is the maximum vertical displacement of the CoM and F_{max} is the maximum vertical force during stance phase. In the second method, the body is modelled as an oscillating object with period T_c and mass m, and Equation 5.14 is re-organised to isolate k_v:

$$k_v = m\left(\frac{2\pi}{T_c}\right)^2 \tag{5.21}$$

The third method is in the same form as Equation 5.12, however the angular velocity of the oscillating body and the body mass are used to calculate stiffness:

$$k_v = m\omega^2 \tag{5.22}$$

The angular velocity is determined from the contact time and the aerial time between successive footfalls on a force plate.

In Butler et al. (2003), leg stiffness is distinguished from vertical stiffness, since the leg contact the ground at an angle and experience a GRF vector at the projected angle. The method used for leg stiffness (k_l) is defined as a function of the maximal force during stance and the change in leg length, measured as the difference in length between two points on the

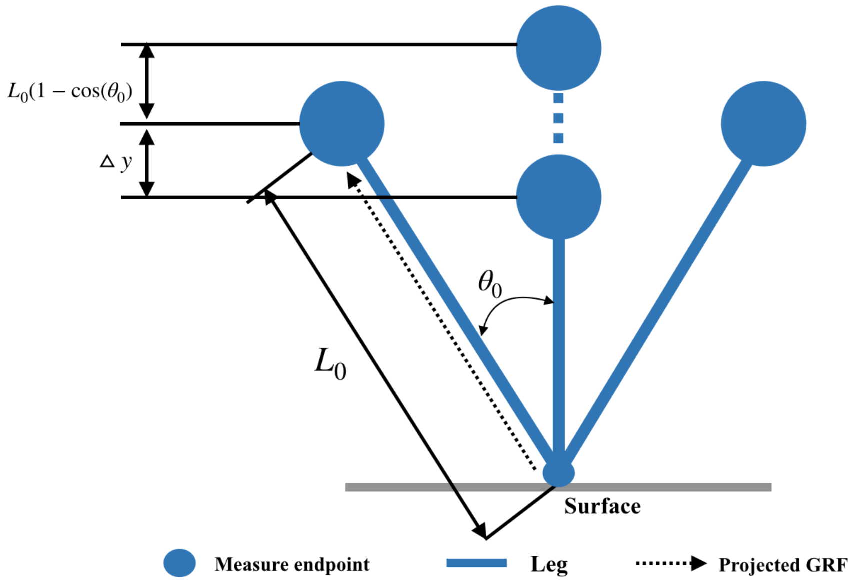


Figure 5.2: Leg stiffness as a measure of projected ground reaction force (adapted from Butler et al. (2003))

leg (see Figure 5.2):

$$k_l = \frac{F_{max}}{\triangle\ L} \tag{5.23}$$

where $\triangle\ L$ is calculated using $\triangle\ y$ (vertical displacement of the proximal point), the leg length in standing, L_0, the half angle of the arc swept by the leg, θ, horizontal velocity u and contact time, t_c:

$$\triangle\ L = \triangle\ y + L_0\,(1 - \cos(\theta)) \tag{5.24}$$

where $\theta = \arcsin\left(\dfrac{ut_c}{2L_0}\right)$. The minimum length of the leg (L_{min}) is reached at the peak GRF, this is when the spring is fully compressed (Figure 5.3).

The third category measures torsional stiffness of joints, k_j, as a function of changes in joint moments (M) and joint angles (θ):

$$k_j = \frac{\triangle\ M}{\triangle\ \theta} \tag{5.25}$$

Using direct 3-D motion analysis, Kulmala et al. (2018) defined leg stiffness as the ratio of leg compression ($\triangle\ L$, the distance between a proximal and distal point of the leg spring) and the projected GRF vector.

$$k_l = \frac{GRF_{proj}}{\triangle\ L} \tag{5.26}$$

Optimal leg stiffness

Per definition, leg stiffness is therefore a reflection of the MSS ability to redirect elastic energy into rotational and linear forward motion with each stride, analogue to the spring that stores elastic energy when its length is changed away from its neutral length. Run coaches such as Bobby McGee (McGee, 2012) and Dr. Mark Cucuzella (Cucuzzella, 2012) term this elastic recoil as ‘free energy’, since the forward momentum of the runner is maintained by elastic return and less active propulsion, instead of energy absorption (or dissipation) and more active propulsion. Put otherwise, the runner should make optimal use of the elastic recoil to propel the body forward, instead of actively pushing themselves of the ground during late stance and toe-off.

However, Butler et al. (2003) accentuates that there exists an optimal leg stiffness for each athlete. Increased leg stiffness (k_l from Equations 5.18) at the same deformation $\triangle\, y$, exposes runners to higher vertical loading forces upon footfall. Higher vGRF loading rates are linked to stress fractures in the lower limbs (Shih et al., 2019) while poor leg stiffness has been linked to soft tissue injuries in the muscles and tendons (Butler et al., 2003). For these reasons, moving away from optimal leg stiffness in either direction, the MSS absorbs the load upon landing through either the bone or soft tissue and dissipates the elastic energy instead of recycling it, predisposing tissues to injuries. The runner, to allow for effective deformation of the spring to capitalise on the return of the elastic energy, should therefore seek optimal leg stiffness.

5.1.5 Recycling elastic potential energy from the springs

For the runner, propulsion forces are a sum of elastic return from the stretch-shortening cycle and activation of muscles. Reduced efficiency of the stretch-shortening cycle demands greater muscle activation to generate upward and forward motion (Moore, 2016). Therefore, the less elastic energy the runner is able to recycle from the spring-mass system, the higher the workload becomes on the muscle to generate the required forces, negatively influencing performance and running economy.

Storage of elastic energy by the stretch-shortening cycle

This spring-like action is described in terms of the stretch-shortening cycle in working muscles, a function of the stretch reflex, consisting of concentric and eccentric muscle activity (Chleboun, 2005). When the muscle has reached its maximum length, shortening of the muscle (concentric contraction) immediately follows elongation (eccentric contraction), although a short delay may be present during a transition phase (Nicol et al., 2006; Groeber et al., 2019). Groeber et al. (2019) reported in a systematic review that mechanical work increased between 9% and 46% during the stretch-shortening cycle when compared to pure concentric contraction. The same review produced studies that showed increases in torque production in the early phase of concentric contraction during the stretch-shortening cycle. The augmented torque was attributed to the return of the elastic energy stored in the stretched muscle.

Elastic mechanical behaviour during the running gait cycle

The stance phase of the running gait cycle consists of mainly two stages: braking (deceleration of the CoM) and propulsion (see Figure 5.3). At footfall, when $\triangle\, y < 0$, (the CoM is moving downward and $y_f < y_0$), the GRF acts on the leg of the runner. In reaction, the muscles contract to decelerate the CoM's downward trajectory during stance and compress the leg-spring. At toe-off, the restoring spring force F_s acts upward to lift the runner into the air (Kulmala et al., 2018).

Figure 5.3 illustrates the compression and release of the leg spring during the stance phase (the leg is now a closed kinetic chain) of the gait cycle. The spring is at neutral length (L_0) at footfall midway through the braking impulse. Maximum compression of the leg (now at L_{min}, modulated through muscle contraction) coincides with the maximum projected GRF at the start of the propulsion phase. At toe-off, the leg spring recoils towards its original length, releasing the stored elastic energy in the muscles, tendons and ligaments (Kulmala et al., 2018).

During ground contact, the lower limbs in stance phase are loaded by the vGRF and the muscles contract (the spring is compressed, that is $y_f < y_0$) and stores elastic energy, E_{pt}. The potential elastic energy is released when the runner enters the flight phase (the applied force is removed and the spring recoils back to its natural, uncompressed length). The elastic energy is

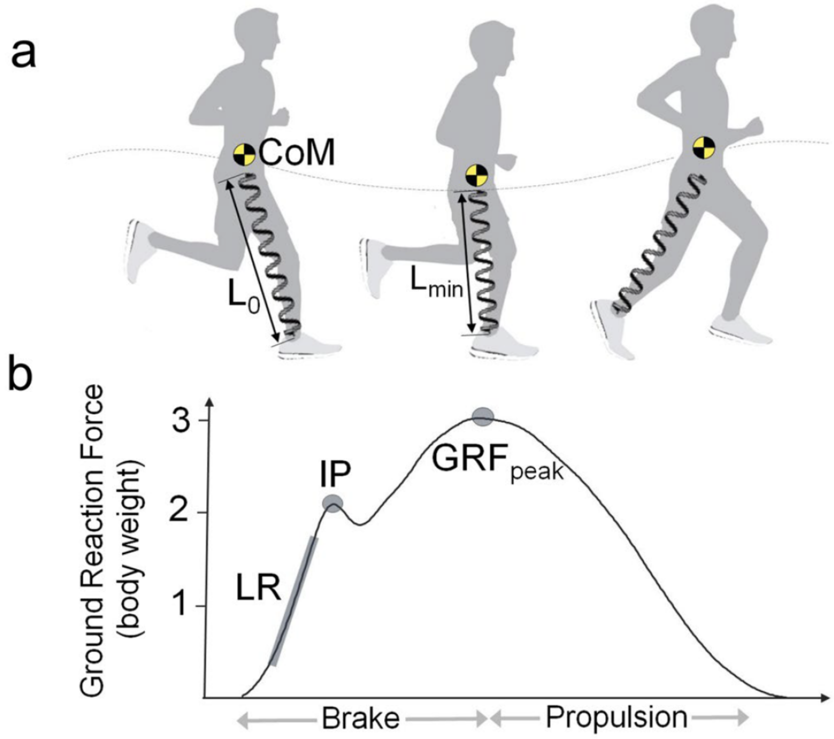


Figure 5.3: Compression of the leg spring and the force-time wave, with permission from Kulmala et al. (2018).

transformed into kinetic energy and the athlete moves through the air over the ground at some velocity v (Morin, 2018; Girard et al., 2013). In this way, positive work was done by the leg to carry the athlete forward over the ground as the linear velocity v increased and the athlete's CoM was lifted to some height h.

The hip drive of the swinging leg is an open kinetic chain. Extension at the hip through concentric action of hip extensors elongates the hip flexors which eccentrically contract; that is, the hip flexors are now in the stretch-shortening cycle and doing negative work, since its final length is greater than its starting length ($x_f > x_i$) (Olney, 2005). The elastic energy is stored in the eccentrically loaded hip flexors (i.e. the spring is stretched out), which will recoil to bring the swinging leg forward (the spring returns to its neutral position). It follows from the studies in Groeber et al. (2019) that greater torque is produced and more mechanical work is done because of the added elastic return, lowering the load on the hip flexors to produce the torque through concentric contraction alone.

5.1.6 The training load function: impulse of the runner's cyclical load function

The runner may be represented as a linear spring-mass system consisting of a single mass (the body) that oscillates around an equilibrium position (defined as where the vGRF equals body weight, (Cavagna et al., 2008)) with the legs acting as springs. To derive the impact force-time waveform for the runner as a spring-mass system, the linear displacement x of the spring from Equation 5.1 is replaced with the time dependent function for displacement, $x(t)$ as a cosine function from Equation 5.5. The vGRF is considered to be an approximation to the spring force in the legs (Ferris and Farley, 1997). The time variable spring force in the legs, $F_s(t)$ as a reaction to the GRFs encountered during running is modelled as:

$$\begin{aligned} F_s &= -kx \\ F_s(t) &= -k_v x_m \cos(\omega t) \\ &= -k_v x_m \cos(2\pi f t) \end{aligned} \tag{5.27}$$

Equation 5.27 is presented as the runner's cyclical load function. Radial velocity, ω is substituted by its constituent variables, $2\pi f$. Equation 5.27 results in smooth, flipped cosine wave (a negative form of the standard wave), without the distinction between initial peak and maximum force characteristic of impact force-time waveform graphs.

The biomechanical parameters for the cyclical load function in Equation 5.27 are usually calculated from GRFs and spatio-temporal variables during force plate and 3-D motion studies (Cavagna et al., 2008; Kulmala et al., 2018; Girard et al., 2013; Clark et al., 2017; Lieberman et al., 2015). In Girard et al. (2013), the stride frequency (cadence), f, was calculated as $1/(t_c + t_a)$ where t_c and t_a are contact and aerial time respectively. Amplitude, x_m, was calculated from the double integration of the vertical acceleration of the CoM (this provided the maximal displacement of the CoM), the original leg length and the angle swept by the leg (the same as per Equation 5.24). The stiffness coefficient of the leg, k_l, was calculated as the ratio between the maximum force occurring at mid-stance and the amplitude. Kulmala et al. (2018) used 3-D motion data (marker trajectories) and GRFs from force plates to calculate position of the CoM, contact time, cadence, and step length. The two-mass model in Clark et al. (2017) only used motion analyses data (from which they derived contact and aerial time and lower limb acceleration) and body mass to model the impact force-time waveforms. Two of the biomechanical parameters for the impact force-time waveforms are available from RWs. Cadence are provided directly, whereas the VO may serve as a proxy for amplitude.

The integral over time of the positive values from Equation 5.27 (that is, the values generated during ground contact) will provide the impulse the runner's body has experienced from its collisions with the ground. Mathematically, the impulse may be integrated over the absolute value of $F_s(t)$, then taking a proportion α associated with ground contact. If the symmetrical split between aerial and ground contact time for lower running speeds in Ferris and Farley (1997) is followed, then $\alpha = 0.5$:

$$\begin{aligned} I &= \alpha \int_{t_i}^{t_f} F_s(t)\ dt \\ &= \alpha \int_{t_i}^{t_f} |-k_v x_m \cos(2\pi f t)|\ dt \end{aligned} \tag{5.28}$$

The total impulse is therefore a function of the 'load-release' cycle that the MSS of the legs as a spring-mass system encounter during running. This impulse in Equation 5.28 is regarded as the training load absorbed by the runner, to which the body's structures must respond. This training load is used as the mechanistic pulsed input function for the simulation model, presented in § 10.3.4 *Selecting training parameters and the main input function: pulsed training load*.

Figure 5.4 presents a simulated waveform of the cyclical load function with vertical stiffness k_v as 20 kN/m, amplitude $x_m = 0.1$ meter and a frequency f of 3 steps per second (values are within ranges reported in Ferris and Farley (1997)). In its current form, the cyclical load function considers the symmetry between aerial and contact time at lower running speeds from Cavagna et al. (2008), therefore the function is evenly split between positive (ground contact)

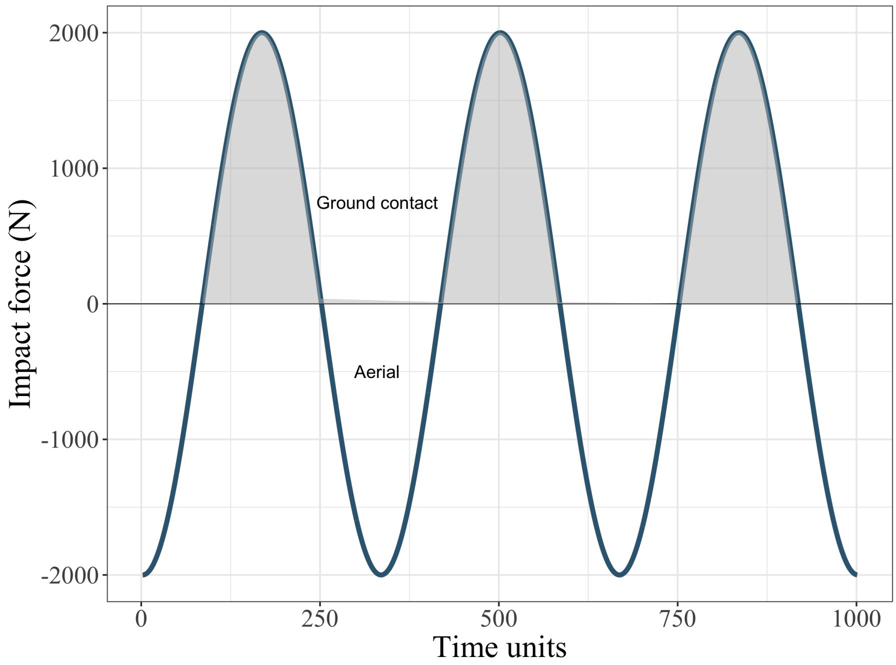


Figure 5.4: Simulated force-time waveform of the cyclical load function

and negative (aerial time) force values. The training load is obtained by integrating for only the ground contact phases, highlighted as the positive area under the curve.

5.2 Entropy and the ambiguity of time in data-driven synthesis of athletic work

The aim of this section is to describe the process as to how training results in momentarily disorder in the body; to which the body responds with adaptation in order to remove entropy and re-establish order and structure. Furthermore, this section highlights the importance of distinguishing the spatial and temporal aspects of the physical activity tracking data from RWs, in order to extract physiological insight. Spatial aspects are related to exposure to variations in the athlete's environment, that is, the surface characteristics as discussed earlier in §4.7 as well as atmospheric characteristics of altitude and temperature. The term *time* in itself is ambiguous, as there are multiple theories and classifications of the concept of the flow of time, both subjective and objective. Two of the objective concepts will be explored in this section: first, time as defined in fundamental sciences of physics, also referred to as Newtonian time, t. Second, the unidirectional arrow of time, T, as emerged from the Second law of Thermodynamics: the idea of entropy, more specifically, entropy as understood in the biological sciences.

5.2.1 Spatial variables and performance

Ambiguity in sport science research is reduced when risk factors are isolated and studies are controlled for by specifying time horizons and restricting spatial variables. Reaching the same type of distinction in the spatio-temporal environment is difficult to achieve in big data from a RW for research purposes, as both the environment (space) and time of recording is out of the researcher's control. Yet, sport science as part of a multi-disciplinary approach to sport research is utilising big data from RWs and online fitness applications to monitor athletes in attempts to understand and perhaps optimise their physical condition (Passfield and Hopker, 2016; Sands

Table 5.1: Proportion (%) of data unsuited for running analyses

Cadence = 0	Cadence ≤ 50	Cadence ≤ 75
6.58	8.78	18.74

et al., 2017; Napier et al., 2017; Kosmidis and Passfield, 2015; Best and Braun, 2017). The real-world data from a RW have other value to offer, if mined correctly: it places the athlete in an ever-changing environment, which can be studied over a longer time horizon. Stated otherwise, the data represents the athlete as an open, biological system: a living organism (the athlete) interacting with its environment to adapt and grow.

A snapshot of a ball rolling on a flat surface will not tell in which direction it is moving: forward or backward? However, when the snapshot is taken with the ball on a slope, a safe assumption (derived from Newton's motion laws) may be made that the ball is rolling towards the ground due to the natural force of gravity. That is to say, it is moving in one direction and cannot move back up the slope in the absence of external work. Placing the ball in another spatial setting immediately provides a different interpretation of its movement in time.

The same may be concluded about an athlete running overground. From the work done by Vermeulen (2018), an homologous view from the geospatial tracking data time-series generated by RWs of an athlete's movement along time did not provide the perspective needed to understand why the athletes are producing a certain running form at some pace. Only once a spatial variable is added, even a qualitative description of their immediate environment, was *changing* performance better understood.

5.2.2 Newtonian time and running biomechanical variables

The data from RWs can be transformed into information regarding an athlete's biomechanics, kinetics and kinematic condition in terms of Newtonian time t, which represents a portion in the universal flow of time when studying a mechanical system. It is assumed that t is reversible and uniform, that is to say that should t be assigned a negative value, the mechanical system's behaviour will simply be reproduced in reverse. Also, all time intervals of $\triangle\ t$ of the same length are identical. A good example is that of a frictionless pendulum: forward and backward motions look the same to the human eye and the same pendulum would behave similar in 1970 and in the present day (Perelman, 1980). Both these assumptions violate what is obvious in the human body's mechanical locomotion system: running forward is much different than running in reverse (or backwards), and a 25 year old athlete running in 1980 will not show the same performance 39 years later in 2019. The time related performance-irreversibility is inadvertently proved with the existence of age categories across sport federations in which athletes of different age ranges may compete against each other. Research on irreversible ageing factors of the human body showed that older adult cohorts have significant lower maximum VO_2, HR, and force production during maximal aerobic exercise testing than younger adult counterparts (Arakelian et al., 2019). Even shorter periods of detraining result in impaired performances in young athletes (14.3 ± 0.7 years), with decreases in aerobic contributions and increased anaerobic lactic contributions in age-group swimmers (Zacca et al., 2019).

Unpublished results obtained by Vermeulen (2018) are considered in this short analysis. A total of 13 494 480 data points from four high calibre athletes, at the same running club, were collected over the four month investigation period. Table 5.1 shows the proportion of data that may (or perhaps should) be disregarded for analyses purposes, as cadences below 75 were considered too low to be representative of running for these athletes.

The data are considered as 'lost', because the true values, as generated during the actual run, remain unknown. It cannot be recovered. The 6.58% lost from a cadence equalling to zero is especially problematic for data validity, as it indicates no movement of the legs, yet speed, change in distances and other data are recorded. It is probable that there must be a number of steps counted after a pre-determined time horizon before the data are recorded as the cadence: $\frac{total\ steps}{\triangle t}$. From this simple equation, it is notable that the time horizon, $\triangle\ t$, plays a pivotal role in the result. It is also possible that a rolling average is used to calculate the cadence, which places even more emphasis on the time horizon. The athlete may have sped up or slowed down onto a different cadence level, but the algorithm for cadence lags behind. The cadence can therefore be inflated or deflated, depending on the acceleration or deceleration of the athlete.

In practice, this means that pace values are being associated with the wrong cadence (Vermeulen, 2018), and subsequently the inference from the data might be negated for monitoring of running form. This simple analyses emphasize the importance of a suitable, yet practical, time horizon $\triangle\ t$ in Newtonian time to measure an irreversible, entropic physiological process (in time T) in changing cadence for the purpose of improvements in running form.

Yet, important running metrics such as average pace ($\frac{\triangle t}{\triangle d}$), average cadence ($\frac{total\ steps}{\triangle t}$) and average heart beat ($\frac{total\ beats}{\triangle t}$) are all calculated in Newtonian time t, with none of them being truly reversible. The data are updated in real-time in intervals of $\triangle\ t$ with a defined length x time units. The interval $\triangle\ t_0$ is not the same as $\triangle\ t_1$, as the two time intervals coincide with a (possibly only slightly) different environment as the athlete moves over the ground. One step might be the difference between running on level terrain and going uphill, which may trigger a change in cadence and running posture (Padulo et al., 2013; Snyder and Farley, 2011).

Monitoring the tracking variables of the runner as a closed system without consideration of his environment only in Newtonian time is a poor indicator of why an athlete got injured, or why they are becoming fitter, slower or faster and why their conditioning change with the flow of time. These variables do not show the physiological changes in the body that take place along another time continuum, T, which is governed by the Second law of Thermodynamics, also referred to as the Law of Entropy.

5.2.3 Entropy of a living system

The fundamental statement of the Second law of Thermodynamics is that transformations taking place in a real system result in qualitative and irrevocable changes in the universe. Time T is unidirectional, evolutionary with processes being irreversible. The degradation brought by heat, friction and other processes of decay occurs only in one way and cannot be revoked. These irrevocable changes are evolutionary in the sense that it has left milestones in T that create history (Perelman, 1980), or present the stages of development of a system. These stages of development provide the directionality time T, in the sense that the future state of a system is not the same as a state in the past. The arrow of time T is therefore unidirectional. A child who has started walking will not spontaneously go back to the development stage of a newborn; neither would a grown tree revert to its earlier state as a sapling.

However, the laws of thermodynamics as defined in physics apply to closed systems, whereas the human body (by extension, the athlete or the runner) is regarded as an open, biological system (Bittencourt et al., 2016; Hulme et al., 2019; Kattman, 2018; Solomon et al., 2002). The definition for the Second Law of Thermodynamics must be re-approached in a biological sense. Locomotion of the human body is a function of mechanical movements of limbs, which in turn is a function of physiology as a branch of biology. Physiology is a function of work in fibres, cells, molecules and so forth in a perpetual cycle of anabolism and catabolism as part of the body's metabolism. Energy is required to facilitate these functions: chemical energy (from fuel provided by the intake of food) is converted into mechanical energy for muscle action on limbs, cardiovascular work to provide blood and oxygen and to remove waste products. From here, the concept of *entropy* and the Second law of Thermodynamics as it applies to biological systems are stated as (Solomon et al., 2002, p. 136):

> *"When energy is converted from one form to another, some usable energy, that is, energy available to do work, is converted into a less usable form, heat, that disperses into the surroundings."*

The dispersed energy is not lost or destroyed, but has been devalued or degraded, as it is more disorganised and diffused than usable energy. This energy becomes unavailable to do work in the living system's body, and thereby the opportunity to use that energy is now irreversibly lost.

Entropy was introduced as a measurement for the proportion of the transformed energy that has become unavailable for work in closed systems. From the dynamical laws of thermodynamics, the entropy in an energetically isolated system, where heat flows from a high temperature body to a low temperature body, will increase until it has reached equilibrium. It will never decrease. This transformational process is therefore irreversible (Loewer, 2012). Entropy has since evolved to describe the degree of order in an open, living system. It may be thought of a measure of energy degradation (or devaluation) during metabolism, disorder and randomness of parts (fibres, cells and molecules) that make up the living system and its sub-systems (musculo-skeletal, cardiovascular, neuromuscular and so forth) and the number of (infinitely) possible states that the particles might find themselves in (Kattman, 2018; Navrátil, 2011; Solomon et al., 2002). The nature of human life processes is irreversible and thermodynamic; therefore, the laws of thermodynamics may apply to human physiology. The concept of entropy may be used to describe life processes in human physiology (Boregowda et al., 2016; Navrátil, 2011; Batato, 1990).

5.2.4 Entropy and the athlete

It follows then, if the human body is considered at first as a closed system where heat is generated inside the body as a result of metabolic activities that cannot be used for other functional work, the body's entropy will always increase in accordance with the Second Law of Thermodynamics. Body structures are therefore subject to irrevocable degradation and decay. How then, if a healthy athlete's body, considered as a highly organised system that is capable of growth and self-repair (to bring itself back from a state of disorder into a state of order (Bittencourt et al., 2016)), can it be subject to the Second law of Thermodynamics with its entropy ever increasing?

Boundaries of the athlete as a living system

The answer lies in re-defining the boundaries of the human body as an open living system. This is provided in Kattman (2018), who mentions three types of open systems: materially open (exchanges of matter), energetically open (exchanges of energy) and entropically open systems. In an entropically open system, living organisms are capable of structural formation by taking in higher order matter or energy (low entropy) with an output of lower order material or energy (high entropy). This intake of low entropy and output of high entropy forms the basis of how the athlete's body interacts with its immediate environment, as well as the universe. The athlete's body requires energy to replace the mechanical energy spent on exertions, but also the heat lost to the surroundings. The key to an ALS's ability to repair and maintain its structures, is to dispose of the excess entropy (heat from metabolic processes and degraded particles from structures that have been broken down during activity) while taking in matter that consists of usable energy, or has low entropy (Kattman, 2018).

Navrátil (2011) concluded with five statements that make up the relationship between entropy and physical quantities in the human as a living system:

1. Entropy of the system increases when internal energy increases.
2. Decay of a system is inevitable unless work is done on the system, either from the surroundings or internally.
3. As time flows, the entropy of the system will increase automatically unless it is supplied with matter of lower entropy.
4. Random activities in a system will increase as entropy increases.
5. Increasing entropy in a system results in less available energy for work.

These statements are considered important in understanding how an athlete's body becomes fitter with better physical performance or weaker, with decreases in performances or injuries that follow suit. Build-up of entropy results in disordered structures prone to random (uncontrolled) activities and in less energy for restorative work. A re-enforcing, degrading cycle develops when the athlete's body is not maintained with the simultaneous supply of low entropy and the disposal of high entropy. Higher entropy means less energy for useful work and increasing random, uncontrolled activities that further break down the structured formation of cells, eventually leading to irrevocable injuries in tissues. For the body to maintain its structural order (fitness) and prevent decay (injuries), work must be done within or on the athlete's body.

The term *negentropy* is the reciprocal variable of entropy: it stands for order, difference or structure (Kattman, 2018). The athlete must take in negentropy in the form of structured energy (nutritional input from carbohydrates, proteins and fats) and engage in physical training, whereby the body's sub-systems develop resilience to countervail the build-up of internal entropy and disorder. Training methods include endurance or aerobic, anaerobic, strength and power, flexibility, speed and agility, skills and cross-training. Each training method has its own purpose by which it improves cardiovascular capacity, and facilitates neuromuscular and musculoskeletal adaptations (Brukner and Khan, 2006) to enhance and maintain structure and order in the athlete's body. Simply put, an athlete's body must maintain an equipoise between negentropy and entropy for it to remain in a healthy, fit condition for optimal performance.

It must be noted that the athlete's body as an entropically open system does not violate the dynamical laws of thermodynamics. When the athlete's entropy is lowered through structure formation to strengthen muscles, improve cardiovascular function or repair injuries with the intake of negentropy, entropy in the universe (his/her immediate environment) must increase when the athlete radiates heat via the skin or excretes other, lower order materials such as water (from sweat) and carbon dioxide during exhalation (Kattman, 2018). Re-drawing the boundary to include the universe and the athlete (the athlete-environment system can now be considered as a closed system), it is clear that the Second law of Thermodynamics holds: entropy will always increase.

Irreversible thermodynamic processes

Borgnakke and Sonntag (2014) provide four factors that render a thermodynamic process as irreversible: unrestrained expansion, mixing of substances, heat transfer through a finite temperature differential, and friction. Three of these factors are now applied to the athlete during running: friction, heat transfer and mixing.

Friction: in physics, to move a body over a surface that is not frictionless from one point to another, a portion of the applied energy will be used to overcome the friction and the rest to move the body. Viscous fluids moving through pipes and channels must also overcome friction. Both these concepts apply to the athlete who is running overground. Friction between the surface and their feet must be overcome during the quick stance phase of the running gait in order to enter the flight phase. The friction encountered by viscous blood in arteries and veins to mobilise nutrients and fuel to muscles and remove waste products such as lactic acid must also be overcome. Energy generated by metabolic activities is required to perform these actions, and internal heat is produced. The system is not returned to its original state, with a raise in internal entropy, which must be discarded to the surroundings for the athlete's bodily system to regain order in its structures so that movement can continue.

Heat transfer through a finite temperature differential: heat spontaneously flow from a high-temperature body to a low-temperature body. As explained earlier, internal heat generated during metabolism is transferred from the athlete's body to the surroundings. Blood flow to the skin during strenuous physical activity may increase more than three-fold in young, healthy individuals (Betts et al., 2013). The surroundings will not be the same after the transfer than before, since its temperature has now increased (perhaps ever so slightly), neither would the athlete's body since it has lost some internal energy that is no longer available for work. The only way to restore the surroundings would be refrigeration of the environment, which requires external work to be done. In areas with a low temperature differential between the athlete's body and the environment (in hotter weather), an athlete might struggle to dispose of the internal entropy as the athlete-environment system is already near equilibrium. Subsequently, athletes may perform worse in hotter environments compared to areas with higher temperature differentials (in colder weather), simply because order cannot be regained in the presence of high internal entropy.

Mixing: blood is a complex liquid composed of water, suspended functional cells, dissolved inorganic substances in ionic form, energy substrates, oxygen and carbon dioxide amongst others (Meyer et al., 2002). Both during rest and physical activity there are constant exchanges of substances between blood and tissues during metabolic activities, breathing and muscle action. However, the processes are accelerated during strenuous training and competing, as energy is required at a higher rate to sustain performance. For example, once oxygenated blood has been de-oxygenated for work in muscle fibres, it cannot be revoked. On a cellular level, carbon dioxide, as a by-product of the energy production process, has been given off to the surrounding blood in exchange for the oxygen, to be transported to the lungs and expelled to the environment as waste (Meyer et al., 2002). This simple exchange resulted in changes in the concentration of oxygen and carbon dioxide in blood as a whole. The contents (or state) of the blood before and after the exchange will not be the same. With continuous training and physical conditioning, the body increases its capacity to use the available blood oxygen (negentropy) effectively and excrete high entropy carbon dioxide. Thereby, despite the irreversibility of concentrations of mixed substances in the blood, the athlete's body maintains its structures.

5.2.5 The entropic line: gauging damage and structural integrity

Earlier in § 4.2 *Typical running related overuse injuries* the terms damage, disorder, disarray, and imbalances were used to describe the aetiology behind RROIs. Injuries represent the ALS or a subsystem being in a poor or non-functioning state due to the cumulative disorder brought on by repetitive loading of unprepared structures. This section introduces the concept of the entropic line as a measure to gauge the degree of additive damage to structural integrity:

$$E_n = \frac{E}{I} \tag{5.29}$$

where E_n represents the net entropy as a unitless *entropy:structure* ratio; E is the amount of internal disorder (entropy) from cumulative micro-damage and I represents integrity of structures. Damage develops to tissues once E_n reaches some physiological limit, that is when the tissue has reached its strain threshold and starts to fail.

Both E and I increase as a result of training, however:

- Entropy, E, is removed through thermoregulation during exercise (to take away excess, unusable heat energy, see § 3.1.4 *Thermoregulation*), cellular repairs to disordered structures, and adaptations of these structures through physiological actions during recovery (that is, to once again form structure that has lower entropy).

- Integrity, I, is only established when the cellular and tissue adaptations have reached maturity. Integrity therefore has a strong, underlying time T component.

An injured runner showing slower performance in Newtonian time might now be considered as a living system of which the internal entropy is increasing. The body is becoming less capable to dispose of the surplus entropy and make use of negentropy (perhaps due to a short supply or a decrease in resilience in structures); thereby E_n increases. I may remain constant at first, but will start to decrease as tissue is broken down and not repaired in time. The athlete's body struggles to regain order in the system to sustain faster paces due to the imbalance between entropy and negentropy and degrading structures.

The increase in E_n is not directly measurable in the time-series data recorded during a run on a RW, but may be captured in some of the running form and biomechanical variables when the athlete is monitored over extended periods of time. Unfavourable changes in variables such as VO, ground contact time (Napier et al., 2017), cadence (Adams et al., 2018; Heiderscheit et al., 2011) and plantar pressure (Wang et al., 2012; Tessutti et al., 2012) may be indicative of disorder in the runner's MSS and/or CVS, compounding internal entropy (increasing E) and further delaying recovery and maturation of tissues (decreasing I).

The environmental stressors from § 4.7 *Environmental stressors in running* influence the entropic line gauge. In a hotter environment, E (heat component) cannot be dissipated at the same rate as in a cold environment; therefore, recovery of order in the systems slows down. At higher altitude, with less negentropy available in the form of oxygen to burn fuel, internal entropy increases (waste components) and structural integrity decreases.

5.3 Conclusion

To summarise, there are specific assumptions on which the eventual systems modelling of the RCAS are based, namely: human physiology is thermodynamic in nature and therefore the concept of entropy applies to athletes; the runner may be modelled as a spring-mass system with elastic behaviour; the runner adjust their biomechanics in reaction to the stiffness of the surface encountered. These assumptions and their links to physics with supporting literature are shown in Table 5.2. The scientific groundwork for the qualitative and quantitative models was laid down in this chapter. The spring-mass system is a well-established method to model the runner as an oscillating system following SHM, with various mathematical definitions of leg stiffness used in research studies. The cyclical load function was presented in Equation 5.27 as the variable impact force experienced by the runner's legs during running. Integrating this function over time gives the training load function (Equation 5.28), which is used as a pulsed mechanistic input to the simulation model in § 10.3.4 *Selecting training parameters and the main input function: pulsed training load*. The research projected presented in this thesis makes use of the cadence as the frequency f of the runner as an oscillator, and assigns various values for vertical leg stiffness (k_v) in the quantitative model to model the cumulative effect of impact loading rates.

An overview of how thermodynamic principles, originally defined for closed systems, are highly relevant to an athlete's physiology was provided in this chapter. The athlete presents a unique kind of open system, namely an entropically open system, capable of structure formation and self-repair with the intake of negentropy and disposal of internal entropy. The link between tracking data from a RW and physiological processes in the human body was made, however analysts must consider the different characteristics of the two concepts of time: the reversible, uniform Newtonian time t and the irreversible arrow of time, T, derived from entropy. The granular, extensive time-series data from RWs should be aggregated to avoid information overload, yet be able to show changes in patterns as *Time* flows. In order for a better understanding in the true conditioning of an athlete as generated in the real world, the aggregated data must be framed to include both temporal and spatial variables.

Table 5.2: Links between running and physics

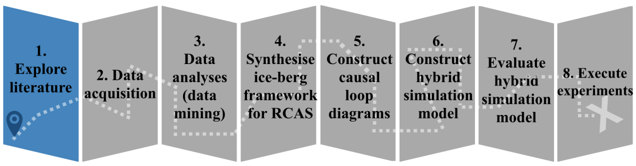
Concept in running Assumptions to model the RCAS	**Physics** Explanation or derivation found in physics	**Supporting literature** Specific sources from where the assumptions were made
Perturbations to homeostasis through an unbalanced, moving MSS; demand for oxygen and fuel from the CVS; thermoregulation.	Entropy (Second Law of Thermodynamics in the biological sense). Human physiology is thermodynamic and the law of entropy applies to humans in their environement.	Perelman (1980); Solomon et al. (2002); Kattman (2018); Boregowda et al. (2016); Navrátil (2011); Batato (1990)
Elastic behaviour of the MSS during overground running	The runner as a point spring-mass system that oscillates around an equilibrium.	Farley and González (1996); Ferris and Farley (1997); Morin (2018); Girard et al. (2013); Moore (2016)
	A spring recycles elastic energy to displace a mass; force in the spring as a function of time t: $F = x_m k \cos(2\pi f t)$.	Halliday et al. (2011)
	The vGRFs encountered by the legs are represented by the force of the spring when compressed or elongated.	Cavagna et al. (2008); Kulmala et al. (2018)
	Runners adapt biomechanics to adjust for surface stiffness	Feehery (1986); Ferris and Farley (1997); Wang et al. (2012)
The training load function pulsed as a cyclical function of time, t: $F_t = x_m k \cos(2\pi f t)$.	Cadence as frequency, f; VO as amplitude, x_m;	Girard et al. (2013) Ferris and Farley (1997); Cavagna et al. (2008)
	Leg stiffness as stiffness coefficient of the spring k_v;	Ferris and Farley (1997); Moore (2016); Butler et al. (2003); Kulmala et al. (2018)

Chapter 6

Methodologies: systems thinking and data-driven computational modelling

Contents

This chapter formulates part of task 1 from the research methodology.



This chapter unpacks ST as a paradigm and a methodology to study complexity. The tools and methods that have been developed to study systems are discussed. Qualitative methods are causal loop modelling, system archetypes, and the ice-berg framework. The quantitative methods are data mining and computational (or simulation) modelling, specifically ABM and SD.

6.1 Definition of systems thinking

Senge (1990) used the analogue of a rainstorm to explain ST. The elements before (amassed clouds, wind, air pressure differentials), during (lightning, thunder, rain, hail, more wind) and after a storm (runoff water, clear skies, ceased wind) are distanced from each other in space and time, yet they are connected in a pattern. The influences of the elements on each other are usually obscured, although their combined behaviour and effects are evident in nature. It is not possible to truly understand the system of a storm by contemplating and analysing the events independently from each other. It is the way in which the elements come together in patterns that constitute the storm system.

As a scientific field of knowledge, ST deals with understanding complexity and change by studying dynamic cause and effect over time (Maani and Cavana, 2007). In ST, the rationale is to study the whole as a synergy of its parts; to see the big picture. As a paradigm, ST is a way to conceptualise the world and relationships, and consists of mainly the following types of thinking (Maani and Cavana, 2007):

1. *forest thinking:* to be able to see both the big picture and how the elements relate and interact.

2. *operational thinking:* the 'physics' of elements in operations and how they affect each other.

3. *dynamic thinking:* the world changes constantly over time.

4. *closed-loop thinking:* cause and effect are non-linear; often the end (the effect) influences the means (the cause).

Richmond (1994) stated that the systems thinker sees both the forest and the trees by having an eye on each. The systems thinker has an appreciation for the details of the elements that make up the system, but is also concerned with the emerging behaviour of the system arising from the interactions between the elements. The emerging properties of the system (forest) cannot be deduced from the original properties of the elements (trees, soil, other plants, sunlight, water) in an additive or summative manner, but rather as the collective outcome from the interactions between the elements (Griffin et al., 2016).

A carefully constructed definition of ST and its main objectives is provided as (Arnold and Wade, 2015, p.7):

> *" ...a set of synergistic analytic skills used to improve the capability of identifying and understanding systems, predicting their behaviours, and devising modifications to them in order to produce desired effects."*

The keywords from this definition are:

- *synergistic*: how things come together to generate combined effects that are greater than the summation of their separate contributions;

- *analytical*: taking things apart to understand the properties of the elements that make up the system.

Synthesis is more than 'putting things back together' after having taken them apart. It is to understand how elements work together (Bartlett, 2001). Working together automatically implies interactions, relationships, feedback. Referring to a storm analogue: a wind blowing in the absence of amassed clouds in sunny weather will not produce a storm on its own; this wind is perceived as a breeze with varying strengths. Rain falling from amassed clouds in the absence of wind is referred to as a shower or a downpour; it does not constitute a storm. Separately the elements that make up a storm system produce other behaviours than what is observed during a storm.

Systems thinking is a scientific thinking framework that make sense of the ever changing behaviour and the complexity of a system as an interconnected whole, rather than an isolated focus on its elements and their functions (Griffin et al., 2016). It is concerned with the interrelationships rather than the elements that make up the system, and to see patterns of change rather than snapshots of system states that are static (Senge, 1990).

However, to build knowledge both analytical (reductionist, linear) thinking and synthetical thinking are required, since the unique characteristics of the parts that make up the system

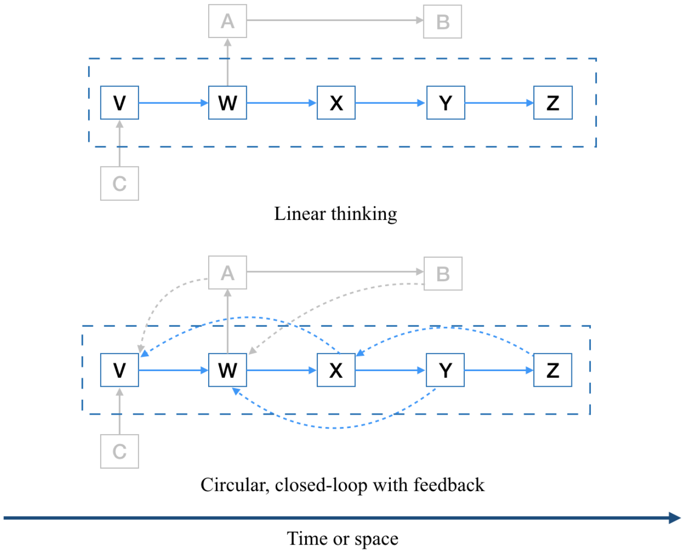


Figure 6.1: Difference between linear and circular, feedback thinking

determine the behaviour of the interactions between the parts. Bartlett (2001) uses the term 'systemic thinking' as an extension of ST to describe the combination of analytical and synthetical thinking: a system is first deconstructed into its elements during analysis, but patterns of behaviour are studied during synthesis. It is necessary to understand how a tree takes up carbon dioxide, sunlight, nutrients and water in order to grow (unique characteristics derived from analyses), whereas the interaction between trees in the competition for sunlight (synthesis of the system to study its behaviour) also affects their growth rate, the positioning of their leaves and characteristics of their trunks.

Closed-loop, dynamic thinking is to recognise that things change with time and that the outcome (effect) may influence the means (cause) (Maani and Cavana, 2007); this feedback process lies at the heart of ST. The complexity of system behaviour is due to the feedback amongst the elements of the system, and not the complexity associated with the elements themselves (Urze and Abreu, 2014). Feedback is the reciprocal flow of information to show how actions may reinforce or balance (counteract) each other. The linear cause-effect principle is replaced with the idea that every action influences another and is itself influenced by some action, that is 'every influence is both cause and effect'. Influence is therefore circular or multi-directional (see Figure 6.1) (Senge, 1990; Maani and Cavana, 2007).

Dynamic complexity evidences itself when the same action elicits different effects in the short and long term (Senge, 1990), which is why ST requires a language or a communication mechanism to deal with not just complexity, but the temporal and spatial separation between elements as well. Systems thinking has its own construct that is uniquely qualified to communicate the interconnected, circular feedback pathways in dynamic systems, where 'x causes y, y influences z and z influences x', instead of the linear language, which stops at 'y causes z' (Goodman, 1991) in Figure 6.1. In a system with expanding boundaries and a multitude of interconnected elements, (now included are A, B and C in Figure 6.1) it is easy to imagine how complex these relationships and feedback structures become.

6.2 Methodological processes in systems thinking

Several methodological processes have been posted in literature to exploit the value of ST to study systems. The general approach is to first construct a qualitative model of the system, which is then translated into a quantifiable dynamic simulation model. The seminal

SD methodology of Anderson and Richardson (1980) has formed the backbone of most ST modelling methodologies. Their methodology consisted of a conceptual and a technical phase. Conceptualisation consists of problem recognition, system conceptualisation and model representation. The technical phase deals with model behaviour, model evaluation, policy analysis and model use. The STMM steps set out by Maani and Cavana (2007) consist of 1) problem structuring, 2) conceptual qualitative modelling (primarily the CLD), 3) dynamic modelling (primarily the SD simulation model), 4) scenario planning, and 5) implementation and organisational learning. However, not all steps nor sub-steps are necessarily required in a STMM intervention. The degree of effort to solve the problem will determine whether all steps are taken. As explained earlier in the research design in Chapter 2, steps 2 and 3 of the STMM inform steps 3 and 4 of the DSRM from Peffers et al. (2008). These steps are summarised as follows:

2. Causal loop modelling. This step adds conceptual rigour and learning power that are inherent to ST and SD.
    (a) Identify the main variables (situations, conditions, decisions or actions that influence other variables and are in turn influenced by other variables in the system).
    (b) Draw the behaviour over time graphs. This includes some quantitative data analyses work.
    (c) Develop the CLD.
    (d) Analyse the loop behaviour over time and typify the loops (reinforcing, balancing).
    (e) Identify system archetypes, if present.
    (f) Identify leverage points, which are the points where applied action may have lasting effects.
    (g) Development of intervention strategies (combinations of leverage actions).
3. Dynamic modelling
    (a) Develop a system's map (or a rich picture). This map or diagram highlights the main sectors, variables and issues of interest of the potential simulation model.
    (b) Define variable types and construct the stock-and-flow diagram (levels, rates, parameters, further explained in § 6.4.2 *The stock-and-flow simulation model*).
    (c) Collect detailed information and data to quantitatively inform the simulation model.
    (d) Create the simulation model from the stock-and-flow diagrams. Assign initial values for stocks and parameterise the relationships of the variables using constants, graphical relationships or mathematical expressions (mechanistic models) in the selected computer package (*Anylogic* for this research project, but many others exist such as *Stella*, *Vensim*, *iThink* and others.) Mechanistic models (for example, the exponential function, cosine functions, and linear regression response functions) may have been identified or formulated during data mining and analyses before constructing the stock-and-flow model.
    (e) Simulate steady-state (or stability conditions). Select the simulation time (hours, days and so forth), the simulation interval (step time, the $\triangle t$) and the time horizon (for how long the simulation must endure).
    (f) Simulate the base case (the reference mode). Compare the output from the base case (the graphs and tabular output) with the behaviour over time analyses.

(g) Verify the parameters, boundaries, and equations used in the model. Validate the model (although validation is not fully possible in simulation models (Sterman, 2000)). The model tests found in Sterman (2000) provide guidance on evaluation of the model: boundary adequacy, structure assessment, dimensional accuracy, parameter assessment, extreme conditions, integration error, behaviour reproduction, behaviour anomaly, family members, surprise behaviour, sensitivity analysis and system improvement.

(h) Do a sensitivity analysis to find key leverage points (or areas of greatest improvements).

(i) Design and analyse policies. Test the policies with the model by changing parameter values, changing the structure of the system or both.

(j) Develop and test strategies (these are combined functional policies).

In this project, not all the steps were necessary.

The constructs and tools used in ST provide a means to understand complexity and dynamic cause and effect (Maani and Cavana, 2007). The ST methodology is concerned with the externalisation of mental models and consists of two main phases: qualitative modelling and quantification of the system. The systems thinker's language of communication is mostly visual, with qualitative modelling tools such as networks, the CLD, structural diagrams, system archetypes (Goodman, 1991), the ice-berg model, rich pictures and cognitive maps (Maani and Cavana, 2007), whereas quantification tools consists of time-series (behaviour over time of system variables), relational graphs, network analysis and computational modelling (simulations or micro-worlds of the real system) such as SD and ABM, Markov chains, machine learning techniques, data and process mining amongst others (Maani and Cavana, 2007; Kirkwood, 2013; Griffin et al., 2016). These tools are related and used iteratively in the modelling process (see Figure 6.2).

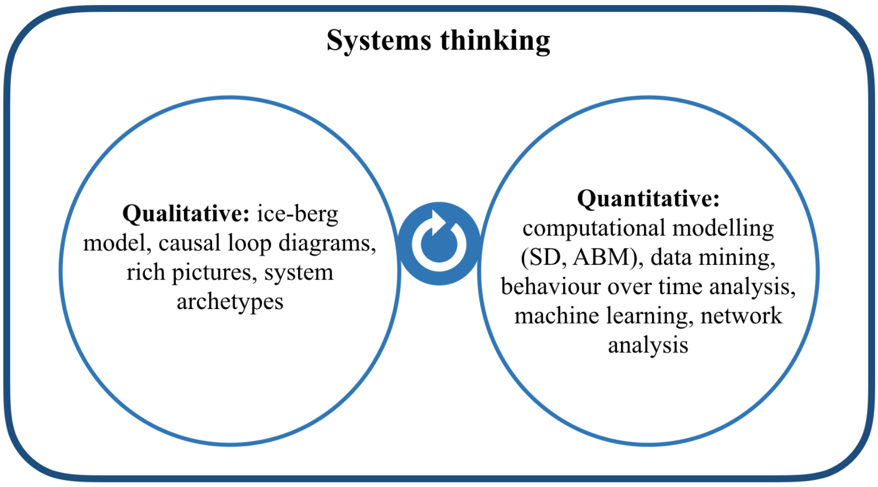


Figure 6.2: Qualitative and quantitative tools used in ST.

6.3 Qualitative tools and methodologies in systems thinking[1]

The focus of ST is not on problem solving *per se*. As opposed to other scientific problem solving methodologies, ST explicitly considers the context of the problem in its environment.

[1]parts of this section were published in *Theoretical Issues in Ergonomics Science*, July 2020

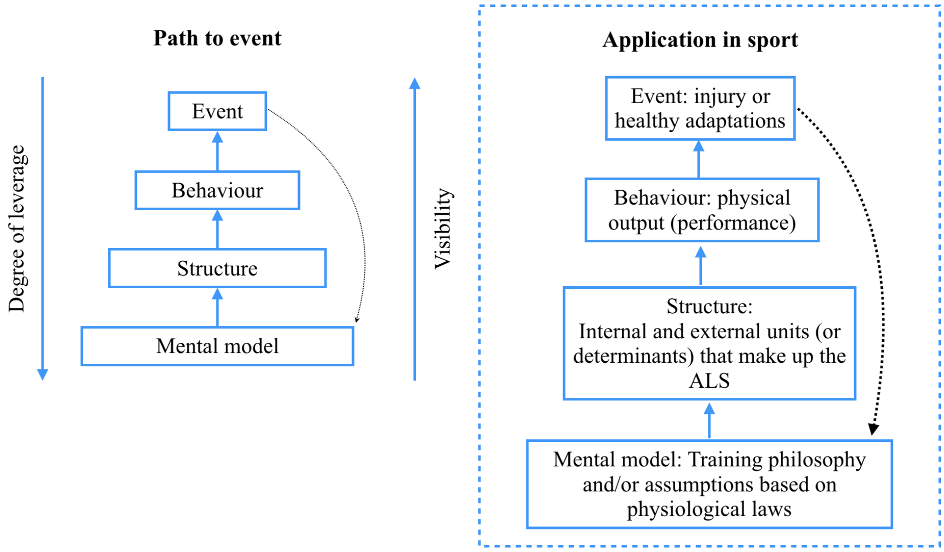


Figure 6.3: Path to event, adapted from Maani and Cavana (2007), Kirkwood (2013). This figure was published in *Theoretical Issues in Ergonomics Science*, July 2020.

Qualitative variables therefore are important factors in a ST approach, since they represent phenomena and/or conditions that are not well accounted for in strict quantitative approaches (Maani and Cavana, 2007). In this section, three qualitative tools that were utilised in this research project are discussed.

6.3.1 The ice-berg model and degree of leverage

Leverage are actions or interventions that may have a lasting impact to reverse trends or break cycles; it requires fundamental changes to the system's structure with much deeper implications than a solution that addresses only symptoms of the problem (Maani and Cavana, 2007). Discerning between high and low leverage for change is possible when the structures that underlie complex situations are visible and understood (Senge, 1990). The ice-berg framework (Figure 6.3, with reference to application in sport) is derived from the hierarchy of thinking based on work by Forrester (1980), Maani and Cavana (2007), and Kirkwood (2013) and facilitates the thinking process behind emergent behaviour: from the mental models to the actual event. The ice-berg framework is used to facilitate the systemic thinking explained by Bartlett (2001): dissecting the complex system of running into its parts and synthesising the system's behaviour through their interactions. The framework highlights the degree of leverage to change the system and the extent of visibility of system behaviour and outcomes (events) for human perception. The structure level of the ice-berg framework consists of the elements and the interactions that make up the system, which are informed by the mental model of the creators as to how and why (referred to the governing laws by Forrester (1980)) the elements interact as they do. The behaviour of the system is a function of the dynamic interactions between elements over time, which eventually give rise to the events at discrete points in time. Feedback (dotted line) from higher levels travels back to the lower levels to alter the input in a bottom-up sequence. The largest amount of change in a system can be achieved when mental models are changed, however this is the most difficult to do as it is the least visible (Maani and Cavana, 2007).

The ice-berg model allows the differentiation of hierarchical system levels in terms of its structure and behaviour, and importantly, how structure relates to behaviour. It is therefore a useful tool to apply in the sports domain to discern behavioural patterns from structural

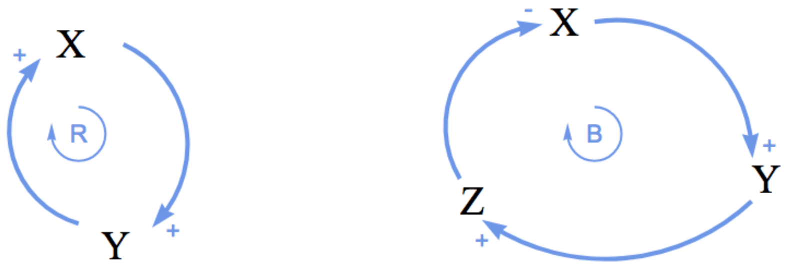


Figure 6.4: Simple CLDs: a reinforcing and a balancing loop

elements, and facilitates the process of identifying optimal and practical leverage. The sports application of the thinking pathway is reflected in Figure 6.3. Training mental models are a function of physiological and physical laws, which govern the ALS and its emergent behaviours. In their complex sport model, Bittencourt et al. (2016) refer to the WoD as the elements (internal biomechanical and external environmental properties) that constitute the athlete's structure as a living system. The interactions and feedback mechanisms within the WoD are mediated by the physiological and physics laws. Although it is impossible to change the fundamental laws of nature's physiology, it is possible to use physiological properties to condition the ALS to develop a favourable WoD and healthy performance. Improved performances or varying injury rates now feed back to the training philosophies' mental models to re-evaluate the approach or affirm the structure of the ALS.

6.3.2 Causal loop diagrams

A CLD presents the qualitative structure of the system with its variables, showing the feedback loops as well as circular causality. There are two basic elements in a CLD: the variables (or factors) and linking arrows. Variables are conditions, situations, actions or decisions that are influenced by, and can influence, other variables. Arrows are polarised links between variables that indicate the causal relationship between two variables, i.e. it reveals the directionality of the relationship: when variable x changes, what happens with variable y? A positive link, represented by a + sign or the letter s, indicates that the variables move in the *same* direction. That is, an increase (decrease) in x would result in an increase (decrease) in y. A negative link, represented by the $-$ sign or the letter o, indicates a reciprocal action, or action in the *opposite* direction. When variable x increases (decreases), then y will decrease (increase). Delays in the system between variables are indicated using double lines on the connecting arrow (Maani and Cavana, 2007).

Feedback loops represent information that travels through the system after some action took place, and eventually finds its path back to its origin where it may influence future actions. Two types of loops exist: a reinforcing loop that reinforces the initial action (also called a positive loop) and a balancing loop that forces the opposite action (a negative loop). The cumulative polarised arrows between the variables determine the nature of the connected loop as balancing or reinforcing (Kirkwood, 2013). These loops later characterise the translated SD simulation model of the system with growth, decline, goal-seeking, equilibrium and stabilising behaviour (SDS, 2018). Figure 6.4 contains two CLDs to illustrate the polarity and feedback loops.

Closed-loop thinking is facilitated through a CLD and is characterised by reinforcing (amplifying), balancing (counteracting) and delayed feedback processes. These feedback structures

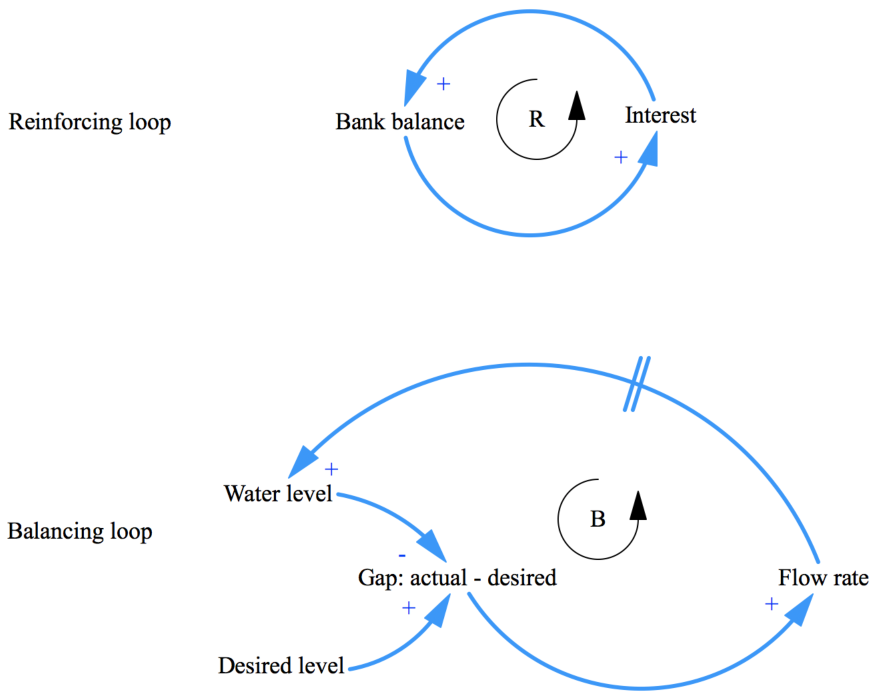


Figure 6.5: Causal feedback loops: reinforcing and balancing CLDs

form the building block of complex dynamic systems. Growth, either positive or declining, is driven by a reinforcing loop (Figure 6.5). Small changes (or actions) amplify into larger consequences over time, that is, movement in one direction results in more of the same movement in the same direction (Maani and Cavana, 2007). Growth can be virtuous, whereby desired outcomes are amplified or vicious when 'things go from bad to worse'. Examples are a growing bank balance due to earning interest as a virtuous loop, or poor performance by an individual that only regresses further due to negative critique as a vicious loop. A balancing loop seeks stability and is driven to close the gap between a desired goal and reality (Figure 6.5). The system will work to produce more of the desired output until the gap has been closed, thereafter it self-corrects in order to stay as close to the goal as possible (Maani and Cavana, 2007; Griffin et al., 2016). For example, the flow rate of a water tank system changes based on the gap between the actual and desired level of water in the tank. When the gap is large, the flow rate is high, but after a delay as the tank fills up and nears capacity, the flow rate should be decreased until the tank is full (Figure 6.5).

Delays are the temporal distance between the actions in a system and the (eventual) consequence (Maani and Cavana, 2007). Every system has inherent delays (Griffin et al., 2016), although some consequences are further removed in time than others: it takes days or weeks for a planted seed to produce a flower; it might take a couple of hours for a small water puddle to evaporate on a sunny day and there are only seconds between opening the faucet and a full glass of water. To recognise and optimally leverage a gap as a result of a delay is key to a system's survival, for growth and stability (Griffin et al., 2016). Overshooting and undershooting goals, unbounded growth and sudden decline and uncontrolled oscillations (when the system cannot reach stability) are the characteristics of system behaviour when delays and gaps are misunderstood or simply (perhaps unintentionally) ignored (Senge, 1990; Kirkwood, 2013; Griffin et al., 2016).

6.3.3 System archetypes

System archetypes represent generic behavioural patterns that recur over and over, thereby simplifying complex system stories into eloquent themes that are easier to recognise. Their structure consists of balancing and reinforcing feedback loops knit together in a CLD with delays to tell the 'bottom-line' plot of the system, by which it highlights low and high leverage for change (Senge, 1990; Peters, 2014; Bureš and Racz, 2016). System archetypes are considered useful as a starting point to system analysis, its synthesis and problem diagnostics (Bureš and Racz, 2016). Several have been recognised and formulated in literature and are explained in much more detail in Senge (1990), Goodman (1991), and Maani and Cavana (2007). The system archetypes are summarised from Senge (1990) and Urze and Abreu (2014) as follows:

- **Limits-to-growth**: A process feeds on itself with a period of accelerating growth. The growth starts to slow down, eventually stopping altogether or perhaps reverse into an accelerated decline.
- **Fix-that-fail**: A problem is solved with the application of some solution that immediately alleviates the symptoms. However, over time, the solution causes unintended side effects, which either reignites the old problem, or develops a new problem elsewhere in the system. Effectively, the original 'fix' had 'failed' to solve the problem.
- **Shifting-the-burden**: An extension of the fix-that-fail. Over-reliance develops on the short-term fix (the symptomatic solution), with the fundamental solution delayed or postponed. With the passage of time, the capabilities of the fundamental solution atrophy or are disabled, prompting more reliance on the symptomatic solution.
- **Drifting goals**: A case of shifting-the-burden, in which instance the symptomatic solution allows for the goals towards which the system strive to decline or erode.
- **Success-to-the-successful**: Two or more actors compete for limited resources or support. Resource allocation is decided upon the degree of success of actors in relation to each other: the more successful actor will gain more of the resource, with the other eventually starved of resources and/or support.
- **Escalation**: Two or more actors view their welfare in relation to one another, acting when the other side is perceived to 'be ahead' in order to re-establish their previously held advantage. This defensive reaction only leads to a build-up (or decline) of assets beyond the desires of all parties involved.
- **Accidental adversaries**: One actor, in a collaborative relationship with another, takes action (which seemed reasonable from their perspective) but in the process undermines the partner's success. A sense of frustration or antipathy may develop between the partners, to the point where they become hostile adversaries.
- **Attractiveness principle**: There are multiple restrictions that limit the growth of activities in a combined process and the attractiveness of each must be managed. It becomes necessary to prioritise the restrictions that must be eliminated.

Two more complex system archetypes are formed as composites of the basic system archetypes, namely the growth-and-underinvestment and tragedy-of-the-commons.

- **Growth-and-underinvestment**: A combination of limits-to-growth and shifting-the-burden. Growth has approached a limit; however, the limit may be eliminated or put off into the future by investment in capacity. The investment must be aggressive and rapid enough to forestall the decline in growth. However, investment is put off by relying on short-term, quick-fix solutions with a more immediate effect to yet again accelerate growth. The system reaches another limit to growth with the repeated use of quick-fixes, and perhaps shifts into negative growth when there is underinvestment in capacity.

In its basic form, the growth-and-underinvestment archetype juxtaposes two solutions to an emergent problem: a symptomatic solution (the quick-fix) that delivers results in the short term, yet are not sustainable, and the long term, fundamental solution which will address the core shortcomings and provide a sustainable solution. The growth-and-underinvestment archetype as spoken in general ST frameworks in industrial processes is outlined with an example of the growth of a business firm from Senge (1990) (Figure 6.6). Product sales in a firm is accelerated (reinforced) in the top-left loop (R) through growing actions, namely aggressive marketing campaigns. However, growth or performance (product sales) to meet demand places a burden on the manufacturing resources, and after a delay, starts to negatively impact the performance standard (as the burden becomes more severe, the manufacturing standards move in the opposite direction), which in turn, leads to poor quality and decreased product output to meet demand (balancing loop B1). Sales have therefore approached a limit due to the burden placed on manufacturing resources by the increases in demand.

Concurrently, a gap opens up between the standard performance (the goal) and the actual manufacturing performance, driving the need to improve capacity (starting the bottom balancing loop, B2). The sales limit may be eliminated or put off into the future by investment in capacity (factories to produce products, skilled staff members and so forth). Newly added online capacity takes away from the burden on resources, resulting in improved performance and meeting production demands (from loop B2 back to loop B1). The investment must be aggressive and rapid enough to forestall the decline in growth. Building the capacity takes time (a delay, indicated by the double lines on the arrow between 'investment' and 'capacity') and directed effort from the firm.

However, investment is put off by relying on short-term, quick-fix solutions with an almost immediate effect to yet again accelerate growth, through more aggressive marketing campaigns. The system reaches another limit to growth with the repeated use of quick-fixes as the market becomes saturated, and perhaps shifts into negative growth when there is underinvestment in capacity, resulting in further degraded quality emerging from a strained manufacturing process. Similarly, insufficient investment in ecological maintenance to support the environment from which socioeconomical systems draw their resources will lead to its degradation and possibly, an ultimate collapse (Bureš and Racz, 2016).

The leverage in the growth-and-underinvestment system is largest when the fundamental solution (B2) is followed (i.e. investment in capacity), with the short lever being the quick-fix growing actions. However, it is possible to leverage the short term solutions in order to maintain output whilst adding capacity.

- **Tragedy-of-the-commons**: A common but limited resource is used by individuals for their needs. Initially they are rewarded with some beneficial return. Over time, as more individuals use the same resource, it becomes depleted with diminishing returns. Individuals then intensify their efforts to increase their return, and by doing so erode the resource even more until it is completely used up.

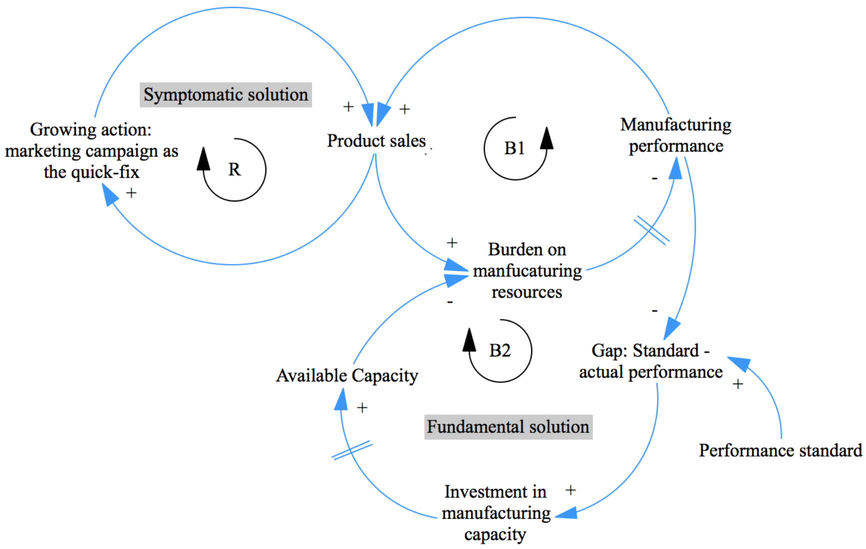


Figure 6.6: The growth-and-underinvestment archetype, adapted from (Senge, 1990). This figure was published in *Theoretical Issues in Ergonomics Science*, July 2020.

6.4 Computational modelling as a quantitative systems thinking tool

Simulation methods to study complex behaviour through mathematical modelling, is an indispensable tool to study healthcare systems (Cassidy et al., 2019). A comprehensive definition of computational modelling is given by the National Institute of Biomedical Imaging and Bioengineering (NIBIB) (NIBIB, 2019):

> *"The use of mathematics, statistics, physics and computer science to study the mechanism and behaviour of complex systems by computer simulation. "*

Such a model has a number of parameters, variables and entities that characterise the system. The model is coded into a computerised simulation, where the parameters are adjusted in order to observe changes in the system's behaviour and eventual outcomes. The simulation model can be used to explore, estimate or forecast system behaviour and events (Koelling and Schwandt, 2005) and is essentially an abstracted representation of reality. The main goal of a simulation model is to develop insight into the behaviour of the system, and not exact, pinpointed predictions of events (Stover, 1980; Featherston and Doolan, 2012; Martin and Schlüter, 2015).

The simulation life cycle starts with the definition of a real world problem that requires solving, alleviation or insight. A conceptual model is developed and coded into computer simulation software, which must be verified that it represents the conceptual model and coding is correct. The conceptual model is an abstraction of reality. Experimentation with various scenarios and parameter variations are performed after the model has been operationally validated. The outcome from these experiments may be used to inform decision makers to deal with the problem or generate better understanding of the real world system (Brailsford et al., 2019).

There are several simulation methods available, but this project focussed on SD and ABM as proposed by Hulme et al. (2019), although in a hybridisation approach. Sections 6.4.1 and 6.4.2 are dedicated to ABM and SD to study systems quantitatively.

6.4.1 Agent-based modelling

Macal and North (2010) explain that ABM is an effective simulation method to model complex systems and a CAS, as it speaks directly to the interconnectedness and their adaptation characteristics. The model consists of autonomous, heterogeneous agents that interact with each other on a low level of granularity (that is, with a high level of detail). The agent acts and makes decisions based on rules (i.e. algorithms) that have been programmed in the agent's make-up. The interactions are the self-organising process between multiple, connected agents and influence their behaviour, which in turn aggregates into the changing behaviour of the system. This is a 'ground-up' or 'bottom-up' modelling approach, whereby patterns, structures and behaviour emerge that was not explicitly coded into the system over time. Agent-based models' parameters and algorithms are based on results from related empirical work. Stochastic sampling from the population adds the realistic uncertainty expected in a system (Auchincloss and Roux, 2008). The modeller can change the input parameters in the agents' and environmental rules as leverage to observe how the system will behave under different scenarios. In this way, an agent-based model can be seen as an algorithm that can study both causal influences and have predictive properties (Macal and North, 2010).

There are two main purposes to ABM: generating hypotheses (similarly, theoretical experimentation or experimental thinking) and prognostic studies (Auchincloss and Roux, 2008) in a safe, virtual environment. These capabilities allows researchers to 'push the boundaries' of research that might otherwise be considered as unethical (pushing an athlete into an injured state) or impractical and costly (instantly replace the stadium's track surface with another material).

In complex sport systems, agents can range from large entities such as sporting bodies, facilities, teams to individual athletes and down to the molecular level of muscle fibres. Agents learn over time based on their experiences, adapt to environmental changes (or changes in other, connected agents) and update internal states (Hulme et al., 2019). Agents change their behaviour, in reaction to the state of the system or encounters with other agents and their environment. The agent-based model may therefore serve to reveal the collective impact of individual actions on the system, and the system's impact on the individual's decisions (Railsback and Grimm, 2011). To explore the mechanisms behind emerging sports phenomena such as rate of injuries on a population level, ABM is therefore a viable option (Hulme et al., 2019).

Agent-based modelling is not the silver bullet that will solve all complex system problems, as it too has some shortcomings. Validation of models is difficult, especially if the system modelled behaved counterintuitively. Errors might slip into the computing code that in turn produces unexpected results (Auchincloss and Roux, 2008). Simulations should be run multiple times and could become a computational burden if the model is not well contained or is too complex for a computer's working memory to deal with, in which case more powerful hardware is required. These challenges set aside, ABM is geared to move complex ST in sport science forward by allowing researchers to expand thinking capabilities into new concepts and untried methods towards improving athletic health and promoting performance.

6.4.2 System dynamics

On the other spectrum of model granularity, SD is useful to study the system's behavioural emergence over time at an aggregated level, or a high level of granularity (Hulme et al., 2019). A critical explication of SD as a discipline is provided here. The reader is referred to the seminal work of Forrester (1961) and Senge (1990), as well as the collection of articles in Legasto et al. (1980). This may be followed up with practical work by Kirkwood (2013) and case studies from SDS (2018) for further reading.

Starr (1980) maintains that SD is a theory of structure, wherein dynamic models of (social) systems may be systematically constructed and analysed. The goal of SD is to understand how the system's behaviour (or performance) is related to its internal structure and operating policies (or governing laws) (Forrester, 1980; Sterman, 2000). A SD model is used to test a dynamic hypothesis: an explanation that provides for the endogenous nature of the dynamic consequences (behaviour) of the system's structure (Sterman, 2000). The focus on the *endogenous* nature of SD is important: it is internal structure, and not external disturbances or influences, which drives system behaviour (Featherston and Doolan, 2012; McGregor et al., 2019).

Leverage for change through policies and interventions

The founder of SD, Prof. JW Forrester, defines policy as an inclusive term for the laws that govern some action. These laws describe the setting of the underlying elements in the structure of the system and how they are connected, by which behaviour is determined (Forrester, 1980). In SD, policy changes encompass the following actions (Starr, 1980; Maani and Cavana, 2007):

- Changing parameter values.
- Changing the linkages between elements.
- Inserting (and removing) certain elements of the system's structure.

By observing the changes on the behaviour of the system under different governing laws, favourable policies may be found to improve the system's structure. The SD tool is aimed at the structural level of thinking and the mental models (refer to Figure 6.3) (Maani and Cavana, 2007; Cassidy et al., 2019). This simulation exercise is perpetual, until the most favourable behaviour and profitable events are found.

The core output from a SD model is to understand the structural sources of the emerging behaviour. From the structure, the key performance indicators can be identified as well as effective policy levers (Koelling and Schwandt, 2005). It is by changing policies and structure that optimal leverage for lasting change or effects may be found (Sterman, 2000; Koelling and Schwandt, 2005). Understanding the causal interdependencies through SD aids decision makers to identify targeted interventions with optimal leverage, and minimises the unintended consequences (Hulme et al., 2019).

The stock-and-flow simulation model

Following the CLD, the computerised SD are constructed as stocks, flows (rates) and tuning parameters that influence stocks and flows (see Figure 6.7). The system process is modelled as a plumbing system, with stocks representing tanks filled with liquid and the flows as the valves (or pumps) that control inter-tank flow. Stocks signify accumulation of a variable (i.e., its level) at any given point in time. Flows are the rates at which the stock level (the variable) changes. Parameters are used to alter the rates. Information on stock levels feeds back into the flows to influence the flow, and in this way the reinforcing and balancing loops are generated (Kirkwood, 2013).

A practical example of a SD model concerning the advertising process is taken from Kirkwood (2013) (Figure 6.8). The CLD presented in Figure 6.8a articulates the process by which `potential customers` are converted through `sales` to `actual customers` during an advertising campaign. The number of `potential customers` increases the number of possible `sales`, and the number of `sales` adds to the number of `actual customers`. This growth is limited by the negative feedback from `sales` to `potential customers`: as `sales` increase, they eventually erode the `potential customers` base, in turn leading to fewer `sales` over time and slower

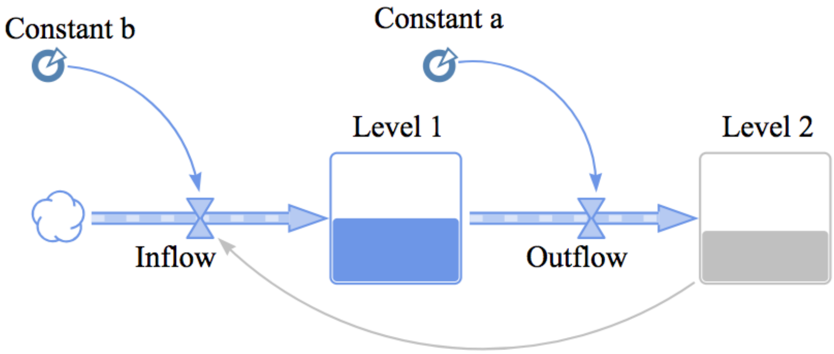


Figure 6.7: Simple stock-and-flow model

growth of the `actual customers` base, until (if possible) all `potential customers` have been converted to `actual customers`.

The stock-and-flow diagram in Figure 6.8b has two stocks, or accumulation variables, representing `potential customers` and `actual customers`. The rate at which `potential customers` move to `actual customers` is presented by the pipe with the valve between the two stocks (the flow), namely `sales`. There is information feedback, presented by the arrow from `potential customers` to `sales`. This arrow implies that the value of `potential customers` somehow influences the value of the `sales`. This feedback was maintained in the CLD of the system: `sales` are negatively influenced (slows down) when the `potential customers` base erodes. The value for `sales` may be represented by a mechanistic model in the form of $\alpha \times$ `potential customers`, where α is a constant proportional rate or percentage.

Functions in system dynamics

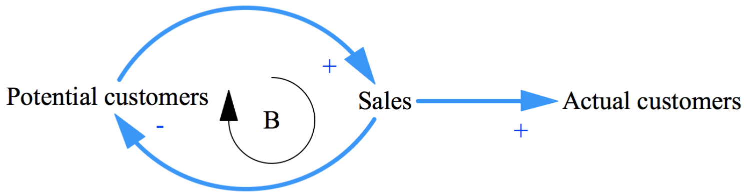
A SD model is, mathematically, a system of non-linear, first order differential and/or integral equations (Cassidy et al., 2019). These equations are coupled. In a simulation, the system of equations is partitioned into discrete time intervals, where the simulation then steps the system through time at a rate of one interval at a time (SDS, 2018). Mathematically, stocks are calculated by means of integrating the net flow rate over the time step $\triangle t$. The flow rates may consist of mechanistic models, such as exponential function and/or waveform functions combined with constants (parameters). In SD, the convention is to represent the integral as a level equation and the net flow rate as r, which has absorbed the constant (Sterman, 2000).

Referring to Figure 6.7, let L_t be the stock level for `Level 1` at time t, $f(t)$ the net flow rate, that is $f(t) =$ `Inflow` $-$ `Outflow` and α a flow rate constant (absorbed constants a and b). Disregard `Level 2` and its feedback to the `Inflow` at first. Then:

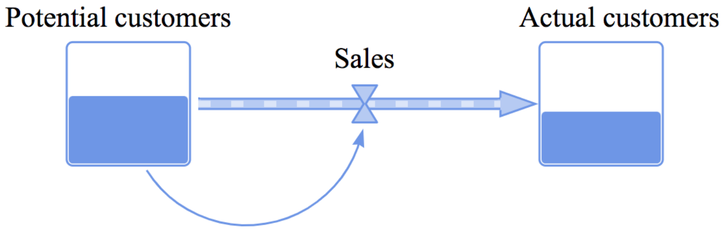
$$\text{Levels:}$$

$$\text{Integral: } L_{t+\triangle t} = \int_{t}^{t+\triangle t} \alpha f(t)dt + L_t$$

$$\text{rewritten as a level equation: } L_{t+\triangle t} = L_t + r(\triangle t) \tag{6.1}$$



(a) Causal loop diagram: advertising, adapted from Kirkwood (2013)



(b) Stock-and-flow diagram: advertising, adapted from Kirkwood (2013)

Figure 6.8: The advertising mechanism

$$\text{Rates:}$$
$$\text{Differential equation: } f(t) = \frac{dL}{dt}$$
$$\text{Rate equation: } r = \alpha \frac{L_{t+\triangle t} - L_t}{\triangle\, t} \tag{6.2}$$

It is easy to see how the feedback from `Level 2` to the inflow of `Level 1` will complicate net flow rate $f(t)$ in a set of static differential equations and integrals. Yet, a computerised SD model is geared to treat such dynamic complexity during the simulation run.

Legasto and Maciariello (1980) provide some guidance on aggregation practices for stocks. Generally, variables may be aggregated in stocks together when:

- They exhibit similar dynamic behaviour, for example physical capital and education.
- They consist of similar dynamic structure, for example capital and technology.

However, variables with different response times should not be aggregated into one variable, for example shifts in population sizes and changes of prices of products.

The delay: a special function

Maani and Cavana (2007) discuss a special function in a SD model, namely the delay, which may be either a material (or physical) delay or information delay. Material delays are the time between the start of an activity and its actual completion (for instance, the time to construct a building). Information delays are lags in the system between updating variables that influence interaction, for example prices and interest rates update slower when smoothing averages are used. The delay implies another level in the system, namely a state of 'being constructed', 'en-route to destination', or 'work-in-progress' (Figure 6.9). In a first-order or exponential delay,

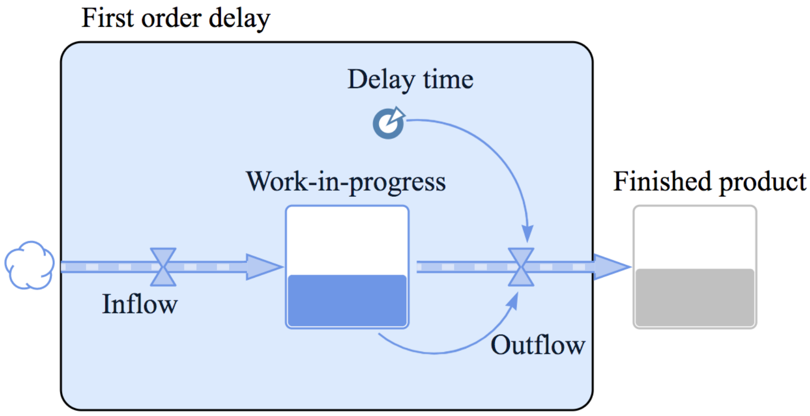


Figure 6.9: A first-order delay stock-and-flow

there would be one more set of inflow, stock (where the material is held up), a delay time and an outflow in the system to represent the delay. Second-order (and higher-order) delays are cascaded, where the outflow from the first delay stock is the inflow to the next delay stock. Mathematically, the outflow (Θ) from the delayed stock is represented as the stock level (L_t) divided by the time of the delay (d):

$$\Theta = \frac{L_t}{d} \tag{6.3}$$

A longer time, d, slows down the outflow and results in accumulation of stock in L_t. In the stock-and-flow diagram (Figure 6.9), the information arrow from the `Work-in-progress` stock to the `Outflow` indicates the dependence of the outflow on the value of the stock and the non-linearity created by the relationship.

Looking ahead to better understand present forces in a system

Both the nature and inevitability of an event are made known by models, but the timing of these events remains uncertain. The selection of a time horizon therefore influences the level of aggregation (Forrester and Senge, 1980; Maani and Cavana, 2007). Forrester (1980) described *"man as a captive of his systems"* (Forrester, 1980, p.16), which by implication means that problems often arise from the very systems created to solve them. Often, the population's needs change with time, sometimes in response to the intervention itself, which cannot be foreseen by decision makers *a priori*. Conventional analytical tools fail to address this dynamic behaviour of the population where disease, risk factors and resources constantly interact and evolve (Homer and Hirsch, 2006). System dynamics is useful in CASs as it is able to purport the unintended consequences of interventions on population level. The reinforcing and balancing feedback loops characterising the problem, might be exacerbated by the intervention itself after some time (Hulme et al., 2019).

Probabilistic future events and tipping points in SD models develop a more complete account of the forces that system behaviour is subject to (Stover, 1980; Featherston and Doolan, 2012). Tipping points in systems, when there occurs a shift in dominance of feedback structures, can be modelled in SD (Featherston and Doolan, 2012). System inertia, or the rate of change in a system, influences the decision for the time horizon for which a model may be useful. Systems with high inertia (slow to change or only change as a result of a high impact force), such as human population systems must be studied on a different time horizon as systems with low inertia (fast to change or changes occur as a result of small changes elsewhere), such as commodity markets (Stover, 1980). The ALS may be described as exhibiting both high and low inertia. High because it takes considerable effort over a prolonged time period to condition the body into a fit state through effective training programmes, low because an injury causes sudden and dramatic changes in the body's structures.

Important considerations: caveats in system dynamics

Parsimony in SD models is salient. Legasto and Maciariello (1980) describes two important aspects during problem definition in SD modelling that challenge its parsimony: system boundaries and choice of an appropriate time horizon. Boundary setting must facilitate all the factors that are considered as relevant to the problem and representative of reality (Sterman, 2000; Maani and Cavana, 2007). When the CLD includes too many elements that verge on irrelevance, the running SD model can become a tedious machine. This is especially true for delayed feedback loops, with third order differentials that exponentially slow computational speed. The CLD and the stock-and-flow version of the SD model must strike a balance to still represent reality, without generating cumbersome calculations that do not add value or insight to the output.

The time horizon of the model must be realistic to capture the system's inertia. A system with high inertia (such as ecosystem and migration studies) will ask for a long time horizon, whereas investment in new products with a lower inertia demands shorter time horizons. The conflict between short-term, intermediate and long-term objectives or benefits from policy changes asks serious ethical and moral questions from the model – the next generation will either benefit or loose from the current generation's system design (Legasto and Maciariello, 1980).

Critique for SD is categorised into four main themes by Featherston and Doolan (2012), namely: the SD model's limited mimicry of the real system, model verification and confidence building, determinism, and tipping points of behaviour. System dynamics have been criticised for models that do not conform to historical system data. However, the purpose of a SD is not to mimic or perfectly imitate the real world, but rather to generate or support understanding of the endogenous structure of the system that leads to its behaviour (Sterman, 2000; Featherston and Doolan, 2012). A SD model must match past patterns of behaviour (such as periods, peaks, trends, amplitudes) but not necessarily specific values at exact time points. In the real world, exogenous factors such as system shocks or other disturbances created data to which the SD does not conform, since these shock or disturbances are not part of the model's endogenous view (Featherston and Doolan, 2012).

As with any simulation or laboratory experiment, modellers must develop confidence in their models: particularly its structure, behaviour and policy implications (Forrester and Senge, 1980; Sterman, 2000; Maani and Cavana, 2007). This is no easy task and may take considerable time and effort, often lending itself into a perpetual cycle without ever realising a useful model (Sterman, 2000). Building confidence in SD models is challenged by the complexity and low visibility of the problems it aim to address (Featherston and Doolan, 2012), which is similar to the technical limitations referred to by Currie et al. (2018) as the uncertainty of causal variables and their values. Yet, McGregor et al. (2019) contend that these uncertainties of variables' values or causality are indications of knowledge gaps that must be filled through other means of enquiry, and that the modelling process of SD is useful in identifying these gaps.

Currie et al. (2018) grouped the limitations and associated challenges for SD modelling as user-related, technical, and application-related. User-related challenges include the need for subject-specific knowledge of the system under study, time investments, difficulty in understanding the complexity of the model, and users' resistance to challenging their mental models. Technical difficulties mostly revolve around the variables of the system and the complexity of their interactions: the uncertainty of causal structures in complex problems, the values of parameters, the data appetite of large endogenous structures, and interpretation bias from subjective variables. Application-related challenges arise from the imbalance of time scales and the goals of the SD modelling process and decision-making: whereas SD seeks long-term leverage, requiring longer time to build a comprehensive model, most public decision making is focussed on immediate and short term outcomes, pressurising modellers to produce over-simplified models.

Modelling the ALS with SD must consider these factors.

6.4.3 Hybrid modelling

In biology, a hybrid differs from its parents but inherited characteristics from both. In simulation modelling, hybrids use combinations of modelling methods to present a new, different method that has inherited the strengths from its 'parents' and reduce the limitations of a single method (Brailsford et al., 2019; Cassidy et al., 2019). A hybrid simulation model combines components of discrete event simulation, SD, and ABM in various degrees of hybridisation (Brailsford et al., 2019). Hybrid simulation allows modellers to deal with different levels of analysis, data availability, data types (continuous or discrete), spatial and temporal scales that are characteristic of complex systems (Brailsford et al., 2019; Martin and Schlüter, 2015; Alvi et al., 2019; Kunc, 2019).

Main application areas for hybrid simulation are healthcare, manufacturing, supply chains, and logistics (Brailsford et al., 2019), social and ecological dynamics (Martin and Schlüter, 2015; Alvi et al., 2019), all complex systems with different levels of interactions, delays and various units of analysis. There are four types of hybridisation in Brailsford et al. (2019):

- *Enriching*, with a dominant method supplemented by minor aspects of another.
- *Sequential*, where the components of the different methods are executed in a pattern (that is, the output from one component serves as input for another).
- *Interaction*, where the execution of components are determined at run time.
- *Integration* are seamless and inseparable models.

In hybrid simulation, both micro- and macro level of analysis is possible. The individual behaviour of humans, animals or other agents that make autonomous decisions in a bottom-up fashion constitute analysis on a micro-level through ABM, but their aggregated behaviour and the feedback within the system on a macro level is more easily studied through SD (Martin and Schlüter, 2015; Alvi et al., 2019). Discrete event simulation conceptualises a system as a network of activities and queues with discrete status updates as entities enter or leave an activity, instead of the continuous changes captured in the stocks and flows of SD (Brailsford et al., 2019). Computationally, micro-level ABM may become expensive for large-scale populations and over longer timeframes, whereas the aggregation methods in SD are meant to deal with larger-scaled systems (Alvi et al., 2019). The different levels of abstraction allow the models to be flexible and enable handling of evolving dynamics in a system (Kunc, 2019).

Data input for hybrid models may be categorised as real world, illustrative, and mixed. Primary and secondary data are real world data, whereas data derived from expert knowledge, hypothetical or synthetic are for illustrative purposes. A hybrid simulation may also use a combination of these types of data in a mixed approach, especially when some data is not available for some aspects of the simulated system and best estimates must be used (Brailsford et al., 2019).

This research project combined ABM and SD interactively to simulate the RCAS on a continuous scale, a special version of the ALS, in Chapter 10. The data sources are mixed: primary (data from RWs and a weather website), secondary (from published literature) and illustrative or hypothetical.

6.5 Data mining in systems thinking

The behaviour over time of system variables are studied mostly by means of time series graphs, but also aggregated in data analyses methods. The data that a system generates as it moves through time is its digital footprint. Data mining can be defined as the process of collecting, cleaning, processing, and analysing data in order to extract knowledge from it (Aggarwal, 2015). It is about discovering useful patterns hidden in raw data by applying intelligent methods to the data (Han et al., 2001). Machine learning is one such intelligent method to perform data operations that will lead to extraction of knowledge.

6.5.1 Driven by (big) data

The big data from RWs are used in this research project to study the runner as a system. Big data in sport are characterised in Vermeulen (2018):

- *Volume:* Data sets are measured in terabytes (where 1 terabyte = 1000 gigabytes) and petabytes (1 petabyte = 1000 terabytes).
- *Velocity:* Fast arrival rates at the computing source (instantaneous in many wearables).
- *Variety:* Data sets can be structured, unstructured, a combination of the two, qualitative or quantitative.
- *Veracity:* Big data arrives with noise from the sensors that are subject to commotion and accompanying errors. This negatively impacts the quality of data streams, and subsequently the validity of the data must be scrutinised. It will be unethical and irresponsible not to refute invalid data, or at least expose it, despite the advances in performance analysis promised by big data (Vermeulen and Yadavalli, 2018).

Variety and volume contribute to the all-inclusive nature of the data, with its low granularity adding to the extensive volume. Some GPS enabled wearables, such as running watches, record data for every second of an activity. For example, 3600 geocodes (latitude and longitude) will be collected during a 30 minute run alone. For further reading, big data is neatly unpacked in Kitchin (2015).

Sports or athletic tracking data sources and/or platforms where data from RWs are stored, that have been mined for research purposes, include *Strava* (Best and Braun, 2017), *Endomondo* (Balaban and Tuncer, 2016), *Sports Tracker* (Oksanen et al., 2015) and *GarminConnect* (Kosmidis and Passfield, 2015; Vermeulen, 2018), amongst others. This project selected the personal run records saved as *.tcx* files of selected runners who partook in Vermeulen (2018), from the *GarminConnect* platform. Environmental, weather related data is available from the *MeteoBlue* website (`https://www.meteoblue.com`) for granular, hourly historic data.

6.5.2 Machine learning for data exploration and clean-up

As systems move through time, their digital footprint may be captured and explained through machine learning algorithms. Within the broader field of data science, machine learning is a branch of artificial intelligence that enables machines to become more skilful in their tasks through intelligent software. The learning takes place through statistical and mathematical methods and models in intelligent software (Mohammed et al., 2016). The machine (that is, the computer) makes decisions, does predictions and forecasts based on data from past experience (Ray, 2019). The machine learning algorithms use data input to build and improve decision-making models and in some cases, execute tasks automatically. The goal of a machine learning algorithm is to define a response function $f(x)$ to predict a dependent variable y from a vector of input variables, $\overline{x}$. Put otherwise, the model finds the optimal set of parameters in $\overline{x}$ to explain y through the response function $f(x)$. For example, algorithms use classification techniques to predict an outcome as a member of a class, or through regression analysis for discrete and continuous response variables (Cust et al., 2019).

Types of machine learning

Three main types of machine learning are characterised in (Mohammed et al., 2016): supervised, unsupervised or semi-supervised, and reinforced learning. Supervised algorithms use labelled data as input to perform classification or regression analysis. Unsupervised learning uses association and clustering algorithms to explore unlabelled data to extract trends and regularities. During reinforced learning, the input is generated by the agent as they interact with their environment and is rewarded or punished upon the output in order to learn correct action (Mohammed et al., 2016).

Algorithms used for supervised learning include linear regression, multivariate regression, logistic regression, random forests, support vector machines, and naïve Bayes, (Ray, 2019; Dey, 2016). Unsupervised algorithms are apriori (Bayesian learning), k-means clustering and principle component analysis (Dey, 2016). Reinforcement learning uses policy optimisation, Q-learning (value optimisation) and evolution strategies (Mehta, 2019).

Machine learning has gained ground in sport science with numerous algorithms put to work in a wide range of sporting codes, ranging from cricket, golf, and football to name a few. The purposes of the machine learning algorithms are diverse, covering aspects regarding training optimisation, body part tracking during execution, ball and/or player tracking on the field, talent identification and career development (Cust et al., 2019; Jauhiainen et al., 2019). In this project, machine learning was used as a data mining tool to explore and clean the data from the RW, specifically those metrics that would serve as input to the simulation model. Clustering and outlier detection algorithms were applied.

Clustering

Cluster analysis is utilised either as a standalone operation to extract knowledge from data, or as part of the pre-processing of data to prepare for use in subsequent algorithms (for instance, in characterisation, subset selection and classification operations) (Han et al., 2001; Ofoghi et al., 2013). The unsupervised k-means clustering is of particular interest in this project during exploratory analysis and preparing the data to sample the values of the input parameters of simulation model.

Clustering partitions a set of data points into subsets. Each subset is referred to as a cluster, of which the points in the cluster are similar to one another, but dissimilar to points from other clusters. A clustering algorithm performs the partitioning, allowing for discovery of hidden groups or patterns in the data (Han et al., 2001). The k-means algorithm clusters a data vector

x consisting of n points into a number k clusters through an iterative relocation heuristic. First, k random centroids from the data vector are selected and the data points are assigned to the nearest centroid to form k clusters. Each centroid is re-calculated based on the mean of the data points within the cluster. The data points in the entire data vector are relocated to their new, closest centroid. This process is repeated until convergence (Shukla and Naganna, 2014), which is when there are no more reassignments of data points to centroids (Han et al., 2001). An objective partitioning function, based on distance between the data points and the centroid, must be optimised by the clusters: the objective function aims for low intercluster similarity, but high intracluster similarity. In this way, points within a cluster are similar to one another but dissimilar to points from another cluster (Han et al., 2001). The objective of the k-means algorithm is to minimise the error $E(S, c)$ in Equation 6.4:

$$E(S, c) = \sum_{j=1}^{k} \sum_{n \in S_j} (x_n - c_j)^2 \tag{6.4}$$

where S is a k-cluster dividing set of entities represented by vectors x containing n data points in the M-dimensional feature space. The non-overlapping clusters S_j each has a centroid in $c_j,\ j \in 1, 2, ...k$ (Kodinariya and Makwana, 2013). Formally, the data set M is partitioned into k clusters $(S_1, S_2, ..., S_k)$ such that $S_j \subset M$ with $S_i \cap S_j = \oslash$ for $(1 \leq i, j \leq k)$ (Han et al., 2001).

In this project, the *kmeans* function in R (R-core, 2020) was used in a multivariate cluster analyses on the input data (cadence, HR and the type of surface on which the run took place) for the simulation model. This function takes as input a numeric vector and iteratively clusters the data based on the number of clusters provided, or use a vector of centroids provided by the user. It minimises the inter-cluster variation (sum of squares between data points and their closest centroid).

Outlier detection

Outlier (or anomaly) detection must find the data instances (or objects) that are dissimilar and/or inconsistent with the rest of the data, exhibiting behaviour that is different from what is expected (Angiulli, 2009; Han et al., 2001). When assumed that some statistical process is responsible for generating the data, an outlier is so different from the rest of the data points that it is suspected it may have been created by a different process (Han et al., 2001). Put otherwise, an outlier is significantly far from its expected position under a hypothesised distribution (Angiulli, 2009). Mechanisms and methods for outlier detection include statistical tests, extreme value analysis, clustering models, distance-based models, density-based models, probabilistic models, and information-theoretic models (Aggarwal, 2015; Han et al., 2001). Generally, outlier detection algorithms serve two types of outputs (Aggarwal, 2015):

1. Real-valued outlier score to quantify the tendency in terms of likelihood or probability that the given point is indeed an outlier.

2. Binary label as a simple, clear indication of a point being an outlier or not. In the end, outlier scores must be converted to a binary label to provide the decision on outlier status.

Several types of outliers are identified in literature, depending on a data point's relationship with the rest of the data: global, contextual and collective outliers. Global outliers (also known as point anomalies) are individual points that deviate significantly from the rest of the data set (Han et al., 2001). Contextual outliers deviate from the rest of the data set when the context of the object is considered; that is, based on behavioural attributes and contextual factors a data point may or may not be an outlier (Han et al., 2001; Malik et al., 2014). An example would be temperature values from given locations and different times of the year. The daytime temperature value of 15°C would be considered an outlier for summer in a particular location, whereas the same value would be normal for winter in the same location.

Collective outliers are those objects that individually are not considered as outliers, but their grouped behaviour deviates from the rest of the data set. Background knowledge about the system and mechanisms of data generation is required to identify collective outliers. The collective behaviour of the grouped individual data points that differs from the rest of the data set may indicate a novelty in the data, or that they were generated by a mechanism different from the one that generated the majority of the data (Han et al., 2001).

During data cleaning, outlier detection and cluster analysis are complimentary to each other, although they serve different objectives (Aggarwal, 2015). In this project, outlier detection by univariate statistical analyses was complimented by multivariate cluster analyses. Outliers were identified through the *scores* function in the *outliers* package in *R*. This function generates normal, t, chi-squared, interquantile range and median absolute deviation scores for each data point and labels the data point as being an outlier or not, based the probability level provided. The chi-squared score is calculated as the squares of differences between values x_i and the mean μ, divided by the variance σ^2 (Komsta, 2015):

$$\chi^2 = \frac{(x_i - \mu)^2}{\sigma^2} \tag{6.5}$$

The multivariate cluster analyses were subsequently used to verify whether the outliers were global or collective. Global outliers were removed from the data set, however the collective outliers were included as a subset.

The black-box problem

Although machine learning is useful in sport decision making, future predictions and may protect against information overload, it suffers from the 'black-box' problem: the algorithm predicts output from data, but does not provide the reasoning or the mechanics as to *why* and *how* the decision was reached (Guidotti et al., 2018). In medical sciences terms, it does not provide the clinical reasoning in reaching its decision. The mental model, or training philosophy in sport from § 6.3.1 *The ice-berg model and degree of leverage* is not challenged, and therefore, if used in isolation, very little to no learning in system leverage takes place. Karkazis and Fishman (2017) raise the ethical concerns when athletic health decisions are made based solely on algorithmic outputs, based on mainly two reasons. These algorithms may be biased towards the creator's mental model of the system, excluding other options or views for the system's structure and behaviour. In addition, the authors question the validity of algorithms, since there is an under-supply of historical, validated data and an oversupply of real-time tracking data used in training and testing algorithms. The data that have been gathered is often considered as comprehensive enough, thereby excluding qualitative factors that cannot be counted or quantified in the same way.

In sport science, both practitioner and athlete insight into the dynamic interactions within the WoD from which physical output arise, is invaluable. Bottom-up algorithms in ABM and SD simulation are transparent, white-box modelling with the purpose to develop insight into system behaviour and find leverage for long-term change. Although white-box machine learning algorithms are developed to address the lack of transparency in most algorithms (Hayashi, 2019), they still do not provide the same extent of user involvement that is inherent to simulation modelling that work towards learning the characteristics of future behaviour, and not only prediction of behaviour. Yet, it is feasible to employ data mining and machine learning to find the most influential variables in the extensively large data sets that should be included in the simulation model, thereby supporting parsimony in the simulation.

6.6 Conclusion

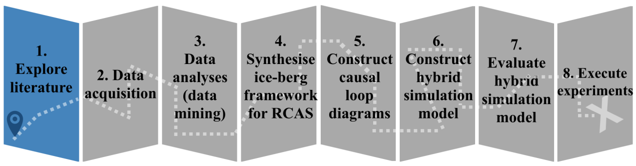
The complexity behind the system's behaviour requires a thinking paradigm to allow for both detail and appreciation of the connected whole. As a structured, scientific approach, ST deals with different levels of detail to study the elements that compose a system and its connected behaviour. Qualitatively, ST identifies leverage for long lasting change. Quantitatively, through data mining and simulation of the system, these leverage points may be trialled and tested. The ST methodology is used to develop insight into the system, with an accompanying simulation model to test intervention policies. The final objective of the ST approach is to develop policies with the greatest leverage for change. The theoretical foundations for both the qualitative and quantitative modelling of the RCAS have been laid down in this chapter.

Chapter 7

Systems thinking and data mining in sport

Contents

This chapter formulates part of task 1 from the research methodology.



This chapter explores applications of the ST paradigm and data mining techniques in sport. Some portions of the work presented here were published in the journal, *Theoretical Issues in Ergonomics Science*.

7.1 Why systems thinking?[1]

Reductionist research approaches have not led to sufficient understanding of the RROI to effectively manage or prevent it (Hulme and Finch, 2015; Bittencourt et al., 2016). Overuse injuries are due to multiple, interacting causes, both internal and external to the athlete, separated through time; for this reason an alternative approach needs to be considered. The techniques from ST may be useful (Bittencourt et al., 2016; Hulme and Finch, 2015; Balaguè et al., 2013; Hulme et al., 2017b; Bekker and Clark, 2016). Living systems have subtle interconnectedness that becomes sensible only through ST (Senge, 1990). From the ST perspective the ALS in all its complexity may be described as unique, complex, dynamic, adaptive, resilient, robust, sensitive, and unpredictable (Senge, 1990; Bittencourt et al., 2016; Sands et al., 2017; Karkazis and Fishman, 2017; Maffetone and Laursen, 2016; Navrátil, 2011), which makes its analysis elusive to traditional linear thinking in scientific analysis.

[1]parts of this section were published in *Theoretical Issues in Ergonomics Science*, July 2020

7.1.1 Complex and adaptive systems

Bittencourt et al. (2016) describes complex systems as dynamic and open systems. They exhibit non-linear behaviour, which is attributed to recursive feedback loops and self-organising interactions between connected elements. In the athlete as a complex system, the pattern that emerges from the self-organisation process gives rise to either an injury or an adaptation in performance. From a system perspective, longevity of the athlete is not a sum of the athlete's functioning parts in isolation; it is the product of synchronised interactions between internal and external elements, which condition and constrain each other towards a common purpose. Advanced statistical modelling, such as found in many multi-factor and data driven research studies, adds to the understanding of the complexity in sport injuries, but do not represent the ST approach (Hulme et al., 2019).

Complex systems in sport range from individual athletes to teams made up of heterogeneous elements (structural and functional) that interact with each other through changing intensities and across separated spatio-temporal scales. The interactions can be mechanical or informal. The system is adaptive and goal directed, with the elements acting as agents, modifying their behaviour as constraints in the system emerge in order to achieve its goal or survive (Balaguè et al., 2013).

In sport, complex systems have multiple, scalable levels, namely: micro (on cellular and molecular level), meso (the individual athlete) and macro (the socio-economic and political facets) (Hulme et al., 2019). From a biological standing, the individual athlete must be considered as a living organism, consisting of multiple subsystems (the organ systems) that work together to maintain life as a whole organism (Solomon et al., 2002). Injuries should be understood at all of these levels. Modulations only at the individual level (for example, through altering the biomechanics of the subsystem, the MSS) do not suffice to prevent RROIs (Hulme et al., 2019). Multiple agents (or entities), which are fundamentally different to each other, interact within and across system levels and over time. The RROI may be attributed to the interactions between the heterogeneous factors of road infrastructure, equipment and technologies (shoes, tracking devices), training pathways, training workloads, biological predispositions, to name a few (Hulme et al., 2019).

As an open system, running does not take place in isolation. Both social (lifestyle preferences, professional obligations, daily stressors) and environmental influences may affect the training quality of an athlete. Research into RROIs should consider scaling up the boundaries of the system under study to include meso and macro level determinants (Hulme et al., 2019), although the congruency and relatedness of the different spatio-temporal scales at which the elements of the system at various levels interact, are important contemplations (Balaguè et al., 2013).

In complex systems, there is a perpetual movement away from stability (Hulme et al., 2019). Adaptations through self-organisation are required to maintain equilibrium (that is, to return to order (Bittencourt et al., 2016)). Runners may be 'pulled' into various directions by innovative products, new sporting events, and peer pressure. In the absence of appropriate adaptations, runners end up in a higher risk state, prone to injury (Hulme et al., 2019). In an unstable environment, athletes may also develop novel, functional actions when the target (or environment) moves stochastically. In this way, when there is interaction between the intrinsic dynamics of the athlete and the external constraints imposed by the environment, there is emergence of individual solutions. The technical skill is used by the athlete to adapt to the changing conditions, instead of producing an ideal motor output (Balaguè et al., 2013).

Taking the idea of complexity and adaptation further, a CAS is a collection of micro-systems that must adapt to the environment for the sake of survival or improvement of the system (Griffin et al., 2016). Examples of a CAS include biological systems, social systems, cyberspace and financial markets. The CAS is a fit description for the athlete who modifies their physical running behaviour when faced with changing terrain, different weather conditions, and other runners' behaviour on the road or reacting to feedback information from their RW. When the athlete is placed in a physical environment, they can be approached as a CAS with many entities that work together as well as against each other and alter the performance of the athlete. These entities are both external and internal to the athlete's body. External entities are the road surface, the geography of the terrain, the weather and many more with the MSS and the CVS as examples of internal entities. Internal interacts with other internal entities, and external interacts with internal entities. From these interactions the athlete's performance emerges as patterns of behaviour in a changing environment, eventually giving rise to a running steady-state, improvements or a RROI.

The athlete and the real world, or their environment, have always existed as a CAS, but has been mostly studied via reductionist approaches under controlled experiments (Bittencourt et al., 2016; Hulme and Finch, 2015; Hulme et al., 2017b). Studies on the environmental effects on athletes performance include work related to the surface (Vermeulen, 2018; Nunome et al., 2017), the gradient (or slope) of the road (Snyder and Farley, 2011; Vernillo et al., 2015, 2016; Vermeulen, 2018), and weather influences (El Helou et al., 2012; Ahmad et al., 2018), all of which come together as the spatio-temporal effects on running.

7.1.2 Taking a systems thinking perspective in sport science

To date, applications of ST in the field are lacking with little practical work to substantiate the approach (Hulme et al., 2018). However, work from Bittencourt et al. (2016) related to the complex model for sports injury, illustrates that through self-organisation (i.e. interactions between the elements that make up the athlete) patterns of behaviour are generated, referred to as the risk or protective profile. Additionally, Hulme et al. (2017b) validated a model of the Australian distance running system to identify opportunities for sustainable intervention strategies towards prevention of RROIs. Through an agent-based model, Hulme et al. (2018) illustrated the quantification of outcomes arising from the complex system consisting of athletes, coaches and training loads that led to injury. Some system archetypes were introduced and practically illustrated in McLean et al. (2019) for recurring issues in football coaching.

Bahr (2016) argues that the current process to screen athletes for risk factors during a periodic health examination, with the end-goal to prevent sport injuries, is insufficient. Intervention studies to support screening for risks to predict injuries are lacking. Screening tests link risk factors with injury in a linear causal pathway, neglecting the different spatio-temporal scales associated with risk determinants found in a pre-season evaluation and eventual injury during the season (Bahr, 2016). Bahr (2016) does not mention ST as a method for risk factor assessment, but does conclude that a multi-factor approach must be undertaken more than once during the season. The case is also made to employ the train-test data methodology, similar to the principles of machine learning, for the validation of screening tests. These suggestions speak to ST in sport science by acknowledging the temporal distance between assessment and injury as well as using multiple factors instead of single factors. Indirectly, the dynamic complexity of the ALS is recognised.

Other work in sport science and running in particular also has an underlying ST approach, yet advances remain lacking. The coaching concept referred to by Barton et al. (2016) based on the work by Davis (2005), ties in with the degree of leverage and feedback in the ice-berg analogue from §6.3.1 *The ice-berg model and degree of leverage*. Running retraining through addressing biomechanical variation is analogous to challenging the training philosophy (the mental model), then altering the athlete's structural and dynamic elements (their biomechanics) in the WoD, and through more favourable interactions emerging from the altered structure the work output by the athlete will be enhanced and the athlete remains healthy. The feedback is circular: this information, the health of the athlete, whose work output is based on the optimal dynamic WoD *for them*, should then return to the training philosophy to either further improve the structural WoD or to maintain it such that the dynamic web may continue to produce the work output. Feedback technologies such as seen in RWs are often utilised to monitor compliance to the altered biomechanics during running (Barton et al., 2016), yet much debate remains to exactly how these technologies must be used for optimal conditioning which is in the best interest of the complex ALS (Karkazis and Fishman, 2017).

Moore et al. (2012) encourage studies to evaluate the interaction between biomechanical variables instead of studying factors in isolation, by referring to the trends seen in opposite sides of the runner's body during running. Both sides of a runner's body undergo adaptations in gait patterns although to a different degree. The magnitude of change might not be the greater stimulus for an economical gait, but how the change affects other biomechanical variables may be of more importance. Although the linear framework in partial least squares regression is useful to deduce influential variables from a large dataset, Trowell et al. (2019) acknowledge that the explanatory variables selected through this technique are not necessarily the only variables that should be considered to understand the relationships between biomechanical variables and running performance. They recognise other contributing factors to variation in technique, such as individual anthropometry.

The RISK (Reduce overall load, Increase capacity to handle load, Share the load, Keep athlete's goals and capacity in mind) framework from Barton (2018) to treat and manage RROIs is edging towards ST, but does not incorporate feedback loops between the RISK components. The framework acknowledges the time delay between injury and return to sport, although still allow for continuation of training and competing despite not being optimally fit by offsetting the re-training to a quieter time in the calendar. This may resemble the growth-and-under investment archetype (refer to § 6.3.3 *System archetypes*): delaying the investment into proper running posture for the sake of continued training and performance may not benefit the athlete in the long term.

Training load has been mentioned as a risk and protective factor for RROI in § 4.3 *Risk and protective factors in running*. Recently, time-varying training load with effect measure modifiers have been promoted in the causality for RROI (Nielsen et al., 2018). These authors suggest the inclusion of training load as the main exposure, but that the exposure be subject to modifiers such as body weight and strength. In this way, other variables (the modifiers) interact with the training load to influence the effect of training on the body's physiological and biomechanical structures. In addition, Nielsen et al. (2019) promotes the time-to-event analysis for sports injuries research, in which time-varying exposure variables of running (such as changes in training load) are the focus. This type of thinking is in alignment with dynamic causal thinking, where variables influence each other over time in a particular direction, but the magnitude and/or intensity of influence changes with time.

7.2 Simulation modelling in sport and healthcare

Computational modelling, and specifically the simulation modelling methods of ABM and SD, is encouraged by Hulme et al. (2019) to study complex systems in sport. The methods of SD and ABM both produce emergent behaviour from designated equations and rules to elements in the system (Hulme and Finch, 2015).

7.2.1 Agent-based modelling

Table 7.1 contains some exemplars of agent-based models to study systems in healthcare and sport, where spatio-temporal interactions between agents and their environments give rise to unique behaviours. Agent-based modelling have been used in problem solving in health systems (Popper et al., 2013) as well as in public healthcare for both infectious diseases and non-communicable diseases (Tracy et al., 2018; Nianogo and Arah, 2015; Zhu et al., 2012).

The two models from Badham et al. (2018) illustrated the usefulness of an agent-based model where the physical environment in which an agent functions have an impact on their behaviour, with the reverse also holding true. These illustrations serve as motivation to employ ABM to study the spatio-temporal effects on running behaviour. The model by Yang and Diez-Roux (2013) fulfils the first purpose of ABM as put by Auchincloss and Roux (2008) as theoretical experimentation, since it is not feasible to change the walking environment to perform walking behaviour experiments. The dynamic, spatio-temporal behaviour of the children and their parents that generated these system level outcomes would not have been possible with methods that are more static such as regression modelling.

7.2.2 System dynamics

Hulme et al. (2019) constructed a proof-of-concept SD model for running injury development on a population level (available online at `https://goo.gl/1fFj3E`). This model accounts for active runners becoming injured, then recover and re-join the running cohort or leave the system. The authors acknowledge that the model may be expanded to include the influence of larger systems on running (social-, healthcare-, economic-, technological, and sports marketing systems), yet the basic form of the model is sufficient to investigate leverage points and policies to alter the running system. The most advantageous cause for SD in sport is to identify the effects of interventions and the reactions of other elements in the system. Some interventions may have undesirable or unforeseen consequences in the future, or the cumulative effects might not be favourable. Understanding the dynamic causal interdependencies moves interventions towards an optimal strategy to minimise injury risks. System dynamics may deal with the complexity of sport injuries in such a way as to translate 'efficacy' of interventions to 'effectiveness' (Hulme et al., 2019).

Table 7.2 contains summarised SD work in sport and healthcare from the literature, each case highlighting an aspect of SD. The model of Dangerfield (1999) underscores the strength of feedback loops, as well as how dominance of loops characterise a system's behaviour. Both Hulme et al. (2019) and Homer and Hirsch (2006) highlight the emergence of unintended consequences from well-intended solutions or interventions, something that might not come intuitively during the design of intervention policies. In Macmillan et al. (2014), the solution in one problem domain contribute to the persistence of another, where two systems interact. The scoping review of Currie et al. (2018) supports the paucity of practical application and reports a low follow-through rate of qualitative modelling to quantified simulation models.

7.2.3 Hybrid modelling

No studies were found for hybrid modelling (SD and ABM) for sport specifically; therefore, the discussion here focuses on healthcare implementations. The systematic review by Cassidy et al. (2019) focussed on simulation modelling in healthcare using SD, ABM or hybrid models. Hybrid models had various purposes, including improving access to care, decrease waiting time, improve patient flow through the system, reduction in undesirable patient outcomes, as well as to estimate future demand. The healthcare settings for these studies are mostly in cardiology, elderly care, maternal care, emergency care, and orthopaedic care. Amongst the rationales for selecting a hybrid approach, are the retention of deterministic and stochastic variability, simulating flow through a system without the need for large scale data collection, individual human variability and detailed interactions are maintained to influence system behaviour, reproduction of detailed, highly granular system elements as well as aggregation of system variables are possible.

Table 7.3 summarises hybrid models as integration examples to study hyperglycemia in pregnancy, safety regulations during construction, and a healthcare technology assessment for a mobile stroke unit. The micro-level interactions are simulated using ABM. Macro or system level dynamics are modelled using SD. Collective output from the agent-based component alter the higher level flows in the SD components, whereas the SD may influence agent behaviour on the micro-level.

Table 7.1: Agent-based modelling in healthcare and sport

Author(s)	**Title**	**Conclusions**
Badham et al. (2018)	Developing agent-based models of complex health behaviour	Incorporation of the time and place effect on health. The TELL ME agent-based model analysed people's protective behaviour during an influenza epidemic. The disease transmission was modelled on a geographic information system's population density data. Mutual interaction between individuals and their environment became visible: agents who were close to new infections adopted protective behaviour and those who adopt protective behaviour reduced the prevalence of the disease in their immediate location.
Badham et al. (2018) (model developed by Yang and Diez-Roux (2013))	Developing agent-based models of complex health behaviour	Generate hypotheses for future research that concerns school placement and safety interventions, where households must decide on their children's commute to school by means of walking. Greater intensive safety improvements close to schools may result in better outcomes towards children's walking commutes than less intensive safety improvements over a large area.
Blok et al. (2018)	The impact of individual and environmental interventions on income inequalities in sports participation: explorations with an agent-based model	ABM served as a proof-of-concept to explore individual and environmental interactions in a complex sport system. Increased sport participation and modest reduction in income inequalities may be facilitated by health education, access (physical and financial) to sport facilities and enhanced safety levels.
Hulme et al. (2018)	Towards a complex systems approach in sports injury research: Simulating running-related injury development with ABM	Introduced the first practical application of an ABM in sports injury science research. From a systems thinker perspective this model demonstrates the dynamic relationships between an athlete's training load adherence, their acute to chronic workload and the RROI incidence on population level.

Table 7.2: System dynamics in sport and healthcare

Author(s)	**Title**	**Conclusions**
Dangerfield (1999)	System Dynamics Applications to European Healthcare Issues	Direct system similarities between diffusion of a new product into the market and the spread of infectious disease. Coupled positive and negative feedback loops (with either one having a characteristic dominance at some point in time) exist in the dynamics behind an infectious epidemic. Quantified SD models can make contributions in epidemiological studies such as done in the Acquired Immune Deficiency Syndrome epidemic.
Hulme et al. (2019)	Computational methods to model complex systems in sports injury research: ABM and SD modelling	Understanding the mechanisms, as well as the timing, of unintended consequences. A new running shoe claims improved shock absorption and foot support. Runners may increase their workload too fast and progress to a RROI.
Homer and Hirsch (2006)	SD Modelling for Public Health: background and opportunities	Unintended consequences of resource allocation in chronic healthcare. Resources were allocated upstream for preventative care and downstream to treat and manage complications. In the long run, the onset prevention resources are absorbed by the demand for downstream care due to complications. Disease prevalence increased, as the onset of the disease could not be prevented effectively while treatment of complications were extended.
Macmillan et al. (2014)	The societal costs and benefits of commuter bicycling: simulating the effects of specific policies using SD modelling	Useful in health impact assessment for cycling as a transport mode. The regional cycle network as planned by Auckland will benefit the city but would not counter the cycling injury rate. Gradual transformation of all roads to incorporate cycling could provide significant contributions towards their transport targets, lower environmental impact and advancement of physical activity of citizens.
Currie et al. (2018)	The application of system dynamics modelling to environmental health decision-making and policy – A scoping review	Five studies (out of 166 articles that were published in the inclusive domain) completed both system analysis and the simulation. An array of policy decisions under study: cardiovascular disease, chronic health disease and associated costs, public health risk factors and health outcomes, malaria control and decision support for responders to extreme heat events.

Table 7.3: Hybrid simulation modelling in healthcare

Author(s)	Title	Conclusions
Freebairn et al. (2020)	'Turning the tide' on hyperglycemia in pregnancy: insights from multiscale dynamic simulation modeling	The tripartite structure of the simulation allowed for multiple levels of abstraction: biological, individual-behaviour and services. ABM components are used for personal characteristics (weight, pregnancy status, age, medical history) while SD is used to model glycemic regulation, impacted by metabolic load due to pregnancy, physical activity, dietary aspects and medication. Discrete event simulation is used for clinical services once diagnosed. A strategy combining targeted intervention and population level promotion of healthy weight through childhood was most effective towards impacting hyperglycemia in pregnancy incidence.
Liang et al. (2018)	Understanding the social contagion effect of safety violations within a construction crew: A hybrid approach using system dynamics and agent-based modeling	Accident preventative measures interact with workers and the environment. Interactions between individual workers and consequences (behaviour that result in accidents or not) are modelled using ABM. On a system level, SD drives information that influences worker decisions regarding safety. The system state may change based on number of accidents due to safety violations by individuals, which in turn will influence the information sent to individual-level decision makers. Intervention strategies must be differentiated based on the hazard situation: low – apply intensive management strategies; high – supplement proactive safety strategies with other interventions such as a high safety goal.
Djanatliev et al. (2012)	Hybrid simulation with loosely coupled system dynamics and agent-based models for Prospective Health Technology Assessments	Persons and innovations (mobile stroke units) are modelled as agents. Population dynamics, disease dynamics and health finances are modelled in SD. Sub-modules are used to model healthcare phases through person state charts (prevention to post-treatment). Higher implementation rates of mobile strike units to deliver thrombolytic therapy resulted in lower disability and savings on long-term care.

7.3 Big data from runner's wearables: a novel data source

Sport analytics as a multi-disciplinary approach to sport research is utilising big data from RWs and online fitness applications to monitor athletes in attempts to optimise their physical condition and keep injuries at bay. Intrinsic risk factors (such as musculoskeletal, cardiovascular, neuromuscular characteristics, and genetic factors) are identified through clinical evaluation by practitioners, although more sophisticated waerables (through specialised sensor technology) will someday provide greater support to the clinical evaluation. The biomechanical and physiological metrics measured by RWs include cadence, ground contact time, VO, HR (Napier et al., 2017; Best and Braun, 2017; Kosmidis and Passfield, 2015), and HRV (Dobbs et al., 2019).

Geographical and environmental parameters accessible from the tracking data include position, distances (through GPS coordinates), pace and altitude (Vermeulen, 2018). Through data mining from wrist worn RWs tracking data, the following external risk factors may be exposed:

- Training errors: excessive volume and/or intensity, inadequate recovery time and rapid increases;
- Surface types (hard, soft, sloped);
- Environmental conditions: hot, cold, humidity (when combined with weather data sets from the local running environment).

Kosmidis and Passfield (2015) found the effective speeds that contributed the most to well-trained athletes' performance during a race, using an analysis from a hybrid set of personal on-field tracking data generated by RWs and physiological laboratory tests. The authors are strong supporters of the application of this data source towards personalised training prescription. Best and Braun (2017) used the tracking data from RWs posted to the online fitness application Strava to explore the differences in HR during mountain and road running. They advocate that this data source provide numerous opportunities for exercise physiology research. Napier et al. (2017) calls for the sport research community to utilise the data provided by RWs.

In support for the data from RWs, Vermeulen (2018) showed that tracking data from these devices might well be used in the future to monitor running form on slopes. Interaction regression models showed sensitivity to changes of the direction of the road's slope, with interference effects from uphill running and downhill running on pace when compared with level running. A runner's biomechanical and performance efforts changed when faced with uneven, sloped terrain. Meta-analysis of the tracking data from fitness applications in urban settings prove that runners change their behaviour when their environment changes, as shown by the visualisation tools created by Balaban and Tuncer (2017) to support urban city planners in their designs of runner-friendly public spaces.

The novelty of the extensive and granular data that are generated by devices such as the RW do present its challenges. These data sets are not validated in the real world and are biased towards those who use them (Vermeulen and Yadavalli, 2018). Still, the potential benefits of the sheer volume of data and its untapped research contributions in sport science (which can perhaps be seen as a proxy to medical science) outweigh the shortcomings. From this vantage, the data sets from a RW provide a starting point into unexplored territory, when its veracity is acknowledged and transparency of methods is maintained. This data source speaks directly to the RCAS.

7.4 Data mining in sport: k-means clustering and outlier detection

Data mining is used to model the interrelationships between performance and attributes, in order to provide support for decision-making (Ofoghi et al., 2013). The aim of conducting a sports performance analysis through data mining methods may be one of or a combination of the following (Ofoghi et al., 2013):

1. Discovering performance patterns with favourable outcomes, through clustering and classification.
2. Predicting performance from prior performance and training measures with relationship modelling and classification.
3. Support for real-time decision-making (what actions to take during the course of an event to optimise the final outcome) based on relationship modelling and rule mining.
4. Demand analysis (what attributes are required to perform the sport at a certain level) through clustering and classification.

Uses for the unsupervised k-means clustering in sport-related research are diverse. Often k-means is a means (data exploration or clean up) to an end (for a classifier algorithm). The k-means algorithm is used in pre-processing of data before classifying shots in the discretisation of gyroscope data for tennis shot timing analysis (Buthe et al., 2016). Xie et al. (2018) employed k-means clustering to minimise errors in a motion state detection based prediction model for moving body parts of volleyball players. A clinical tool (the Kerlan-Jobe orthopedic clinic shoulder and elbow score) to diagnose shoulder injuries is evaluated for specificity and sensitivity using k-means clustering (Gaudet et al., 2019). The k-means algorithm may also be incorporated as the end in itself, such as cluster analysis of sports participation profiles for the European Union, to derive data-driven policy decisions regarding health promotion strategies through sport participation (Van Tuyckom, 2013). Dingenen et al. (2020) classified two subgroups of runners based on the k-means clustering of lower limb kinematics. They make an important statement: *"the same running-related injury can be represented by different kinematic presentations, and similar kinematic presentations can be related to different running-related injuries"* (Dingenen et al., 2020, p.105). Their findings may assist in the clinical reasoning towards a diagnosis and treatment plan of an injured runner, yet practitioners should guard against the bias that a certain running form or pattern of movement generates specific types of injuries. Table 7.4 contains examples where the k-means clustering was part of the classification process.

Sport specific applications of outlier and/or anomaly detection in athletic (or other physiologically) generated datasets may focus on either cleaning the data from outliers, or finding outliers as the purpose of the operation (see Table 7.5). O' Donoghue et al. (2015) propose a multi-step anomaly detection process in the time-series analysis of HR and distances covered by soccer players: boundary detection (similar to contextual outliers), univariate outlier detection using statistical parameters and multivariate principle component classification. Jauhiainen et al. (2019) approached talent identification as an anomaly detection problem, citing imbalanced datasets due to the rarity of elite athletes. They used a support vector machine classifier to detect talented young soccer players, based on physical tests, technical skills, speed, and agility of players as anomalies to the normal performance of their peers. Louzada et al. (2016) used the z-score ($z = {}^{(x_i - \mu)}/_{\sigma}$) in detecting outliers of general scores computed from principal component and factor analysis in real time for soccer talent identification.

Table 7.4: K-means clustering in sport

Author(s)	**Title**	**Conclusions**
Buthe et al. (2016)	A wearable sensing system for timing analysis in tennis	The inertial measurement unit's gyroscopic data from tennis rackets are used in the longest common subsequence to classify tennis shots. The data is first discretised through k-means clustering, which provided the similarity score of the time series segments that belong to a certain class. Subtle differences in shot execution by the player influenced classification, but with a user-dependent approach classification accuracy was brought up to 94%.
Van Tuyckom (2013)	Six sporting worlds. A cluster analysis of sports participation in the EU-25	A hierarchical and k-means cluster analysis on data from the Eurobarometer 62.0: Standard European Trend Questions and Sport (across organisational context and sport participation intensity). Six disparate clusters were identified, ranging from none-to-average fitness with participation in commercial, organised contexts to very active with a wide range of sporting participation context (organised and non-organised). The analysis support the notion that intervention and sports promotion strategies must be varied across the clusters. Clustering is dynamic, therefore the methodology may be well suited to monitor European sporting participation and inequalities over time.
Dingenen et al. (2020)	Subclassification of recreational runners with a running-related injury based on running kinematics evaluated with marker-based two-dimensional video analysis	A group of 53 recreational runners were classified into two homogeneous subgroups, based on the k-means clustering of kinematic data of the lower limb complex (hips, knees, and feet). The significant differences between the groups included greater foot inclination and tibia inclination at foot strike for subgroup 1. There was a tendency towards lower limb (shin) injuries for subgroup 1, and hip injuries for subgroup 2. Injuries at the knee and foot are more evenly dispersed. Similar kinematic patterns are linked to various RROIs and the same RROI may be represented by various kinematic patterns.

Table 7.5: Outlier detection in sports data

Author(s)	Title	Conclusions
Lin and Chen (2015)	A study of efficiency monitoring systems for match-fixing players in the Chinese Professional Baseball League	Fraudulent play is approached as an anomaly to the players' normal efficiency rate. Efficiency changes were evaluated using a weighted moving average. Detection accuracy improved to 100% for fielders involved in match-fixing scandals in 2008.
Louzada et al. (2016)	iSports: A web-oriented expert system for talent identification in soccer	A general score is calculated from a physical test variables (relative power, VO_2-max, and 20m cyclic speed) and a technical variables (kick, dribble and pass) using principle component analysis and factor analysis. Each individual player's score is measured against the average of the group to determine how far the player is distinguished from others through a z-score. That is, an outlier (statistically different from the majority), based on the z-score, would be considered an above average player.
Jauhiainen et al. (2019)	Talent identification in soccer using a one-class support vector machine	Four different datasets of soccer players were used to train a one-class SVM anomaly detection method. Physical tests performance datasets with a large sample size resulted in better AUC-ROC values ($0.763(\pm 0.007)$) than smaller scaled questionnaire data ($0.585(\pm 0.062)$). It is suggested that talent identification may be more efficient when using non-linear methods than linear methods, including larger number of variables and players.

7.5 Conclusion

This chapter reviewed some practical applications of ST and data mining in sport and healthcare domains. The novelty of the ST approach in sport science is reflected in the paucity of practical work to substantiate the approach. Systems thinking and the accompanying quantitative modelling may contribute to both prevention and management of RROIs on various levels of interventions. Systems thinking approaches are supplementary to reductionist approaches.

Part II

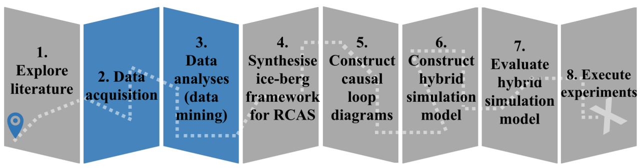
Model development

Chapter 8

Data analyses: the digital footprint from the runner's wearable and the weather pattern

Contents

This chapter formulates tasks 2 and 3 from the research methodology.



The RCAS is described through data mining processes to present the athlete's digital footprint obtained from the RW. This footprint is vitally important as it serves to parameterise the simulation model of the RCAS in Chapter 10. The most important features of the analysis are the interaction between the surface type and cadence and between HR and cadence. Both time-series and aggregate data models are based on the data representing a moving athlete, that is, instances where a cadence of greater than zero were recorded. The data presented here originated from athlete 3. The other athletes' graphs may be found in Appendix A.

8.1 Input data management

This study followed the same raw data extraction from the RWs, processing and cleaning procedures as found in Vermeulen (2018) with some added features explained in the following sections.

Running metric data from the runner's wearables

A total of 3034.2 km of running data (consisting of 58 240 320 unique observations across eight variables) were processed from the participating athlete over the period from January 2017 up to August 2019. The running data were aggregated on two levels: per-run (or session) and subset per run (or surface) type. The per-run aggregation is used in the behaviour-over-time analyses of running metrics (§ 8.2), and to calculate the probability mass function (PMF) for the run type (surface) distribution as input into the simulation model. The per-surface type aggregation is used to inform the parameters in the simulation model (§ 10.3.10) and for general statistical modelling (distributions, histograms, ANOVAs, and boxplots) in § 8.2. The different run types represent the dominating surface encountered during the run, namely road running and racing (asphalt and concrete), trail running and racing (grass and gravel) and track running (grass).

Data were cleaned during processing on separate occasions:

1. The digital footprint, for the behaviour-over-time analyses, was cleaned to include only recording of a moving athlete, that is, for cadences greater than zero.

2. A contextual clean-up to limit the HR to a physiological maximum and to include only instances of running (distinguished by a transition speed of 2 m/s, (Neptune and Sasaki, 2005)) for realistic input into the simulation model.

3. A distribution sensitive clean-up to stabilise the input for the simulation model.

Weather data

Historic weather data from the *MeteoBlue* website were extracted bi-monthly and added into an *SQLite* database. The data were aggregated to a monthly level, per location and only for the early morning hours (05:00 to 08:00) and late afternoon to evening hours (16:00 to 20:00) to stabilise the temperature range. The averages, standard deviations, minimums and maximums of temperature were extracted as parameters to inform the *Anylogic* simulation model (§ 10.3.10).

Simulation input data clean-ups

The final range selected for the input data (for cadence and HR) resulted from a combination of contextual removals during multivariate analysis and outlier detection using the data distributions. The underlying data were also tested for normality. After contextual outliers were removed, the data were subjected to test for global outliers using the *scores* and *outliers* functionality in *R*. Within the *outliers* function, the data points were scored against the *chi-square* distribution at a probability level of 0.95 for outliers. Those points detected beyond the 0.95 probability level were regarded as outliers. The *chi-square* distribution was selected to maintain the values in the upper tails of the distributions. Under the *normal* distribution these values were regarded as outliers, however they should be considered since they were generated by different mechanisms (mostly track running).

The distributions of cadence and HR with outlier detection limits are shown in Figure 8.1. The wide dispersion of cadence is apparent when visualised with distinction of outlier status. The range of cadence (82 to 98) within limits is much smaller than the total range. However, iterative cluster analyses (§ 8.3) revealed that the higher cadences (above 95 strides per minute) are attributed to track running. These observations are therefore considered as collective outliers, since they were generated by a different mechanism (middle distance running and/or short distance sprinting) as opposed to long distance running. Therefore, the lower limit of 82 was applied in the distribution based clean-up of cadence data together with the 99th percentile as the upper-limit for cadence. The higher cadences were included in the input as a subset of track running.

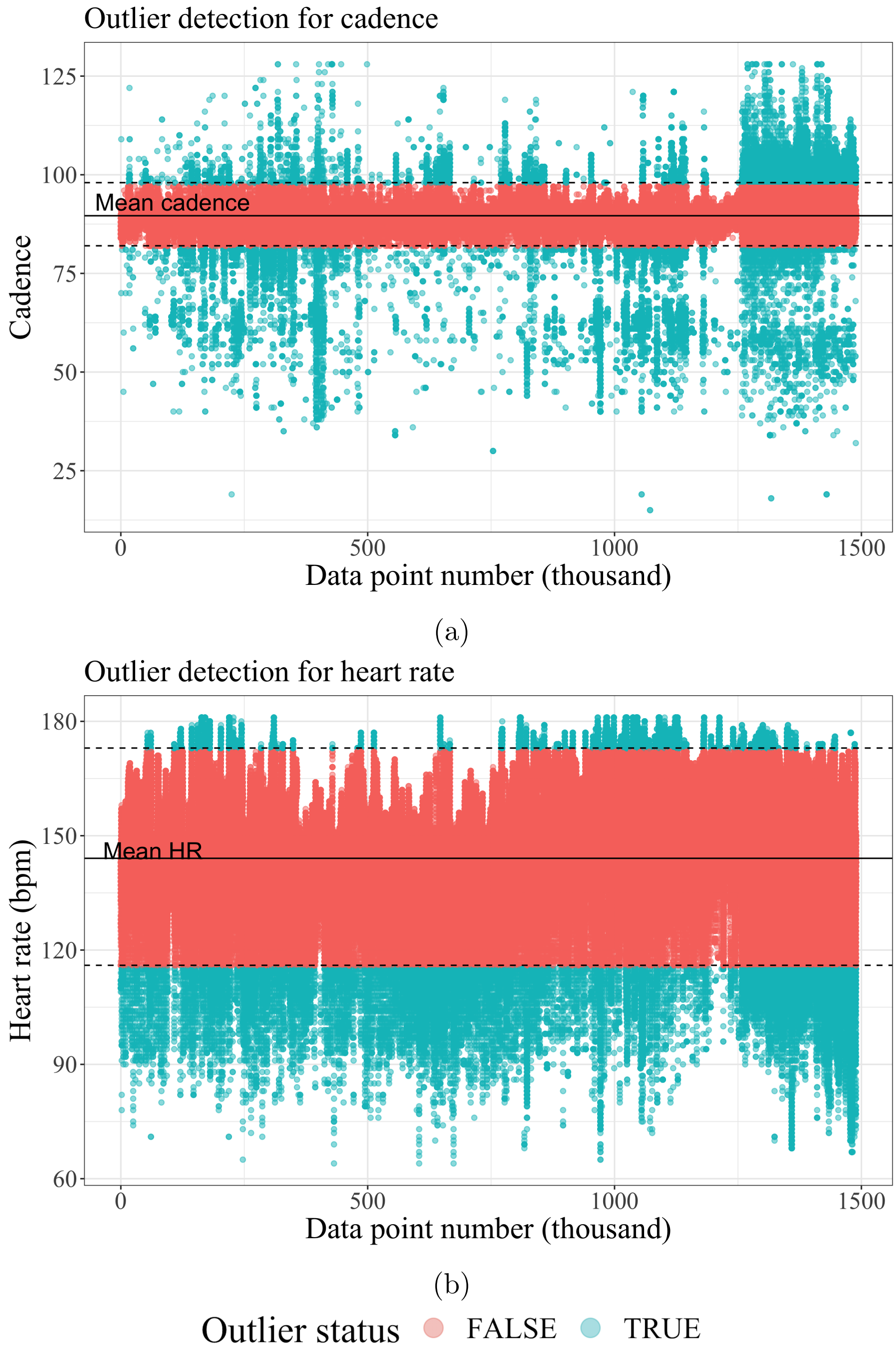


Figure 8.1: Distribution based outlier detection for cadence and HR

The simulation is performed only for the geographic region surrounding Pretoria, South Africa. The large majority of the athlete's runs took place here. Subsequently, the data for Pretoria and surrounds were extracted based on the latitude and longitude values. The *k-means* clustering algorithm was utilised to better understand general behaviour and distribution of cadence and HR for the location-specific data. The clustering served as a guideline to confirm that reasonable sampling parameters are provided for the simulation model. The final input values for cadence and HR for the simulation model were sampled from the cleaned data and checked for consistency with the cluster analyses to be representative of the distribution.

Table 8.1: General statistical measures of dispersion for running data

Metric	Mean ± st.dev	Range (min, max)	IQR (25^{th}–75^{th})
Heart rate (*bpm*)	144.71 ± 13.16	116, 172	135–155
Cadence (strides per min)	89.69 ± 3.22	82, 102	88–91
Pace (*min/km*)	5.20 ± 1.02	0.00, 8.30	4.60–5.75
Distance (*km*)	9.95 ± 6.27	0.15, 53.75	6.27–11.02

Table 8.2: Frequency for run types

Run (surface) type	Frequency
Road race (rc)	4
Road running (rr)	289
Trail race (tc)	13
Trail run (tr)	76
Track training (tt)	152
Total runs	534

8.2 General distributions and behaviour over time analyses

General statistical distribution parameters for HR, cadence (strides per minute) and pace are shown in Table 8.1. The time-series and interactions between the runner's biomechanics, physiology, and the environment are modelled on the per run-session aggregated data set (from § 8.1). The time-series behaviour is subset into the different running types as a proxy for the surface, namely road running and racing (asphalt and concrete), trail running and racing (grass and gravel) and track running (grass). Frequency of run (surface) types is shown in Table 8.2. The athlete participated mostly in road running, followed by track running and trail runs.

Heart rate

The histograms for HR across running (or surface) types (Figure 8.2) reflect the random distribution seen in Figure 8.4a, with the mean for both road and trail racing shifted towards the right. Road running has the most symmetrical distribution. Trail running tails to the left and track running has a tail to the right.

One-way ANOVA tests for HR showed statistically significant differences between the means when differentiated by running type groups (*p-value* $< 2 \times 10^{-16}$). Differences between groups were found to be significant in the Tukey honest significant differences test (on a 95% confidence interval) that followed the one-way ANOVA. Figure 8.3 show the boxplots for HR subset by running type.

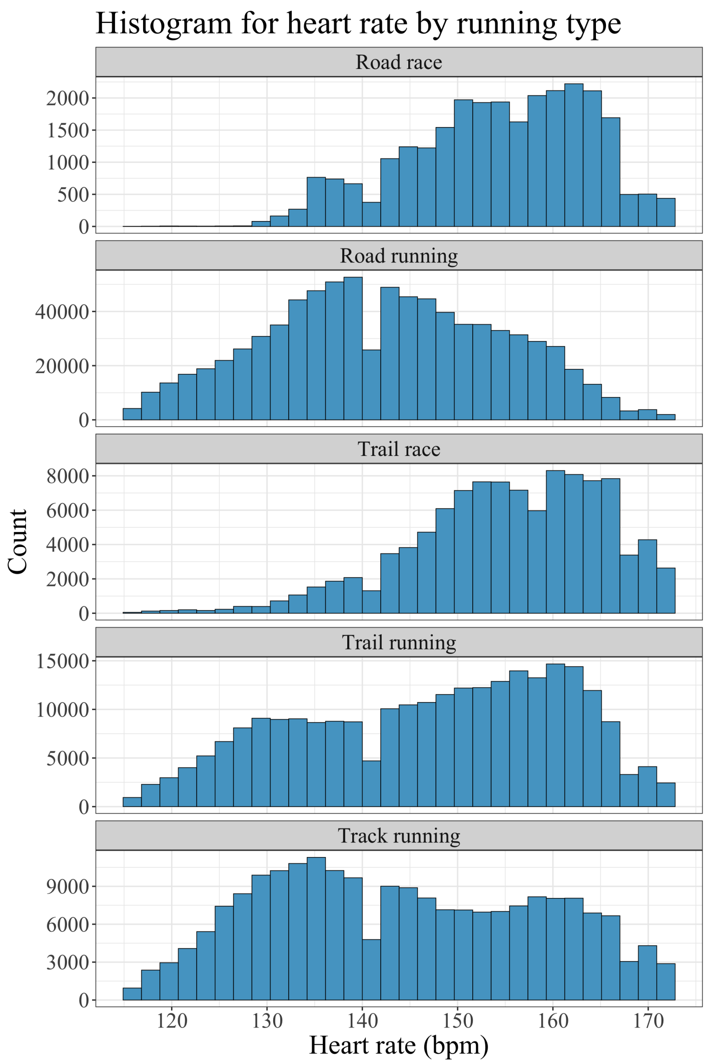


Figure 8.2: Spread of heart rate across run (surface) types

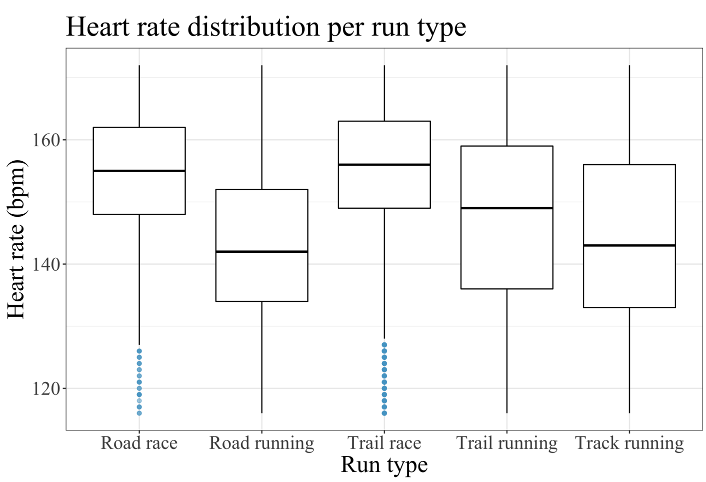
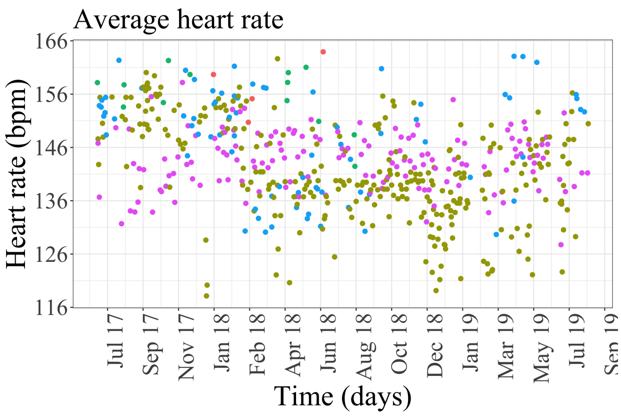
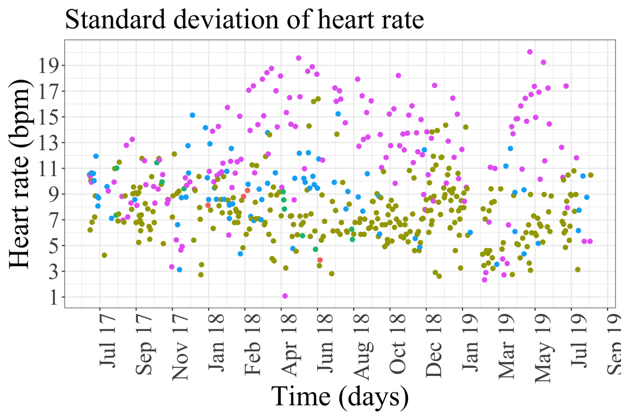


Figure 8.3: Boxplots for heart rate, per run (surface) type

Discussion of the per-run aggregated analyses of the HR data starts top-left in Figure 8.4. The average HR for the time period (Figure 8.4a) seems to be randomly distributed for all the running types. There is a difference in the standard deviation of HR (Figure 8.4b), with track running consistently being higher than the other running types. The average HR for morning and afternoon (or early evening) runs in Figure 8.4c seem to differ in the range, with morning runs being more dispersed than afternoon runs. The interaction between HR, distance and running type seems to show a funnelling pattern for increasing distances, that is, HR tends to some optimal level within the random dispersion with increasing distances (Figure 8.4d). Average HR across altitude is shown in Figure 8.4e. Generally, the athlete's HR is lower at lower altitude, in agreement with the physiology explained in McArdle et al. (2010).



(a)



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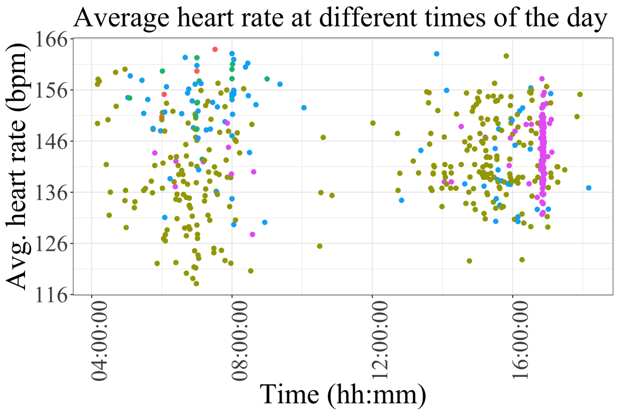

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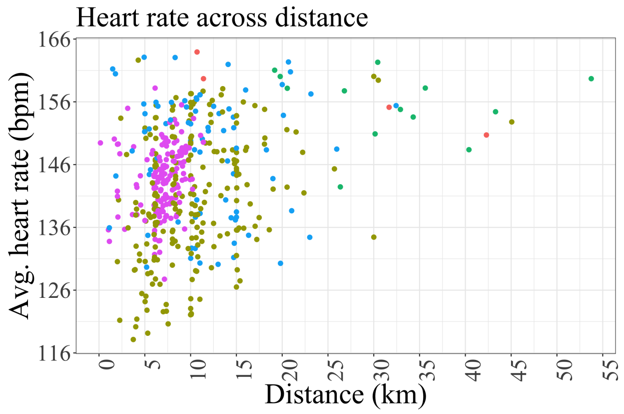

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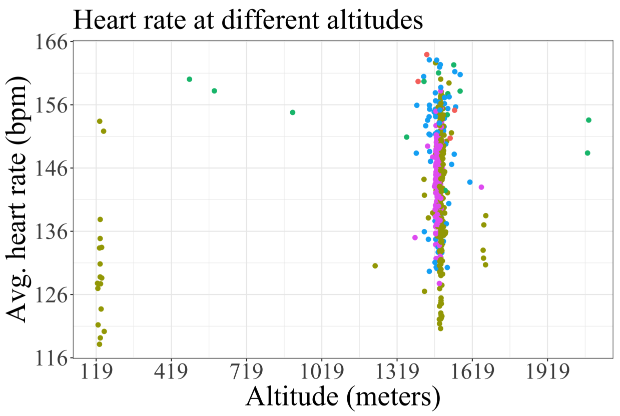


(e)

Figure 8.4: Behaviour of heart rate

Cadence

A multivariate analysis for cadence (here in strides per minute) and surface type in Figure 8.5 shows the bi-modality of the cadence distribution for track running, with road racing also presenting a secondary, minor peak. Cadence peaks between 88 and 90 strides per minute for all the run types. Concerning the main peaks, the histograms for cadence is near symmetrically normal for road running, trail running and trail racing. Track running has two separate peaks at 88 and 97 strides per minute. One-way ANOVA tests for cadence showed statistically significant (though, in some cases small) differences between the means when differentiated by running type groups ($p\text{-}value < 2{\times}10^{-16}$). Differences between groups were found to be significant in the Tukey honest significant differences test (on a 95% confidence interval) that followed the one-way ANOVA. Figure 8.6 show the boxplots for cadence, subset by running type. The differences observed between the groups are small in some comparisons, yet statistically significant.

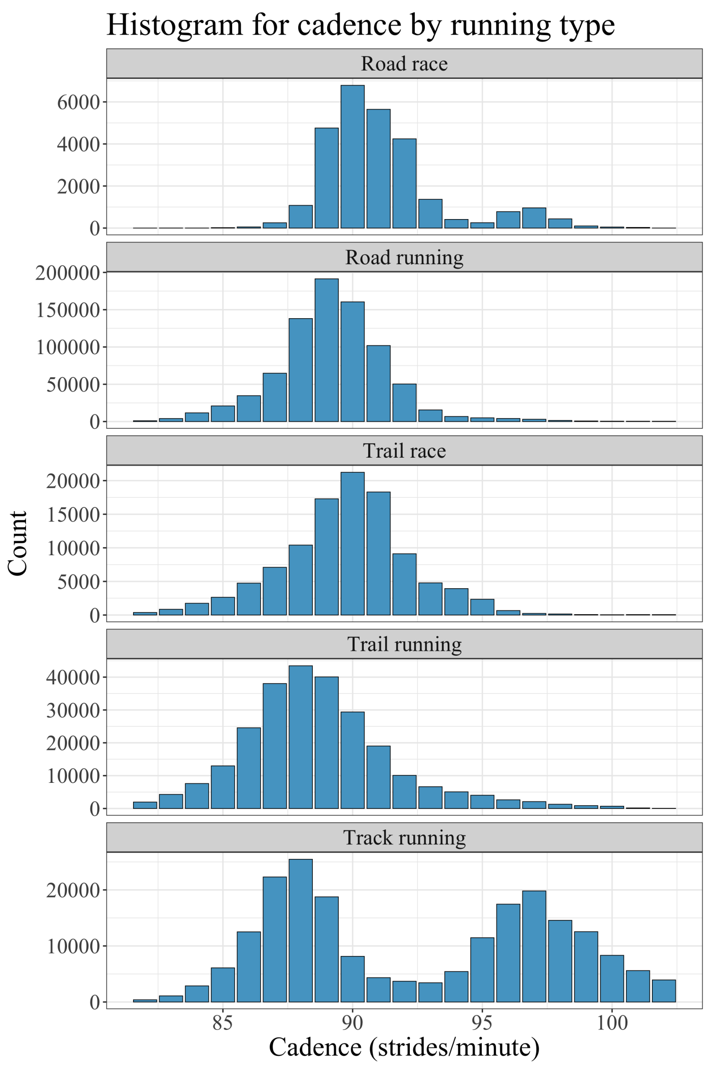


Figure 8.5: Histogram for cadence, per run (surface) type

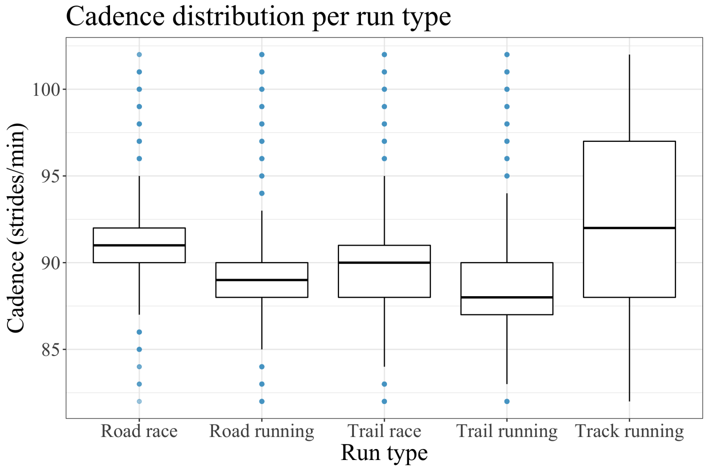


Figure 8.6: Boxplots for cadence, per run (surface) type

Figure 8.7 shows the behaviour of cadence over time, as well as the interaction HR and distance. The funnelling pattern is more pronounced for average cadence in Figure 8.7c. The relationship between HR and cadence is shown in Figure 8.7d. The points seem to be randomly distributed, although a general upward trend (higher HR for higher cadence) is visible. This is consistent with literature: a higher cadence places a larger demand on circulation to achieve a faster radial velocity of the lower limbs (Snyder and Farley, 2011).

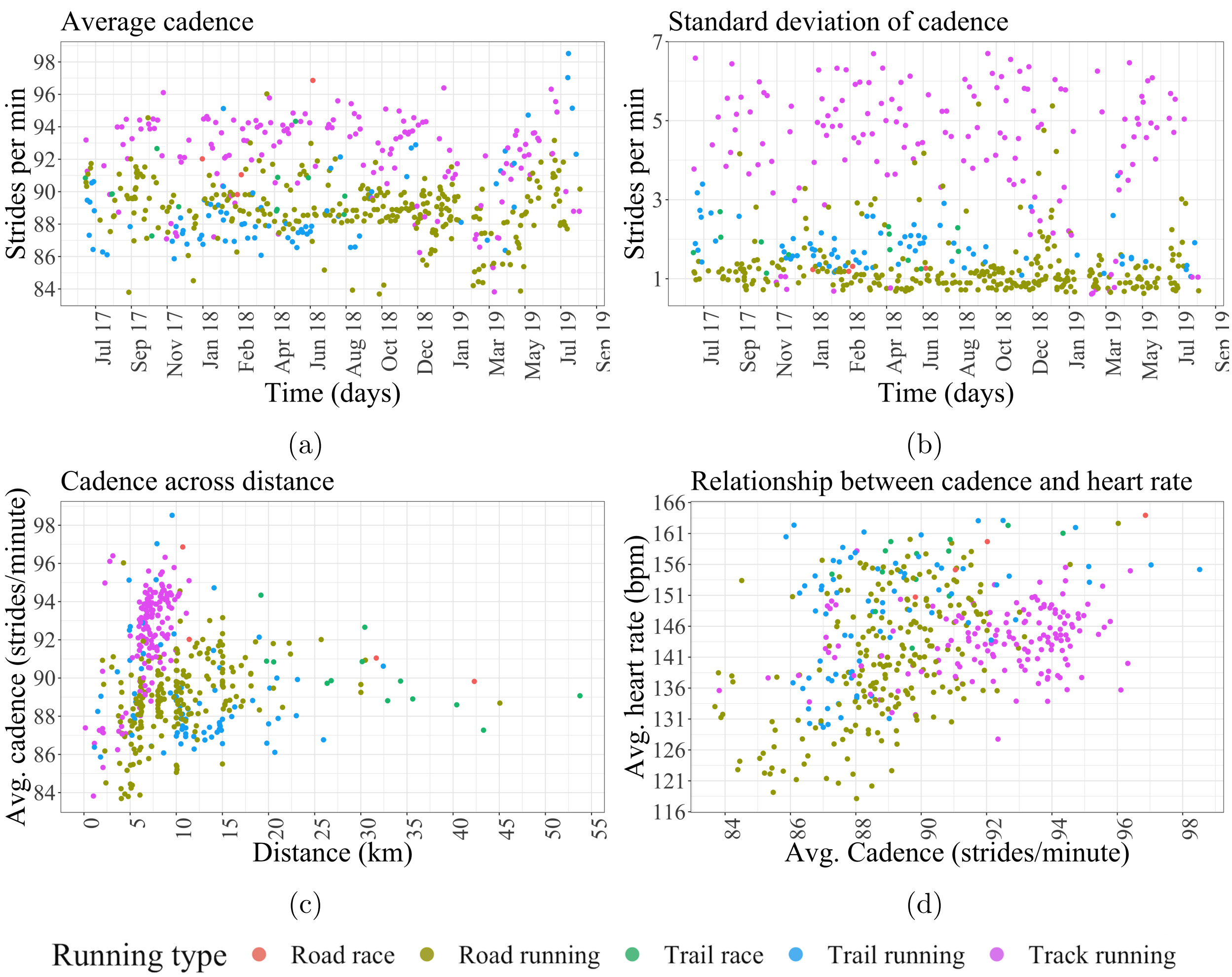
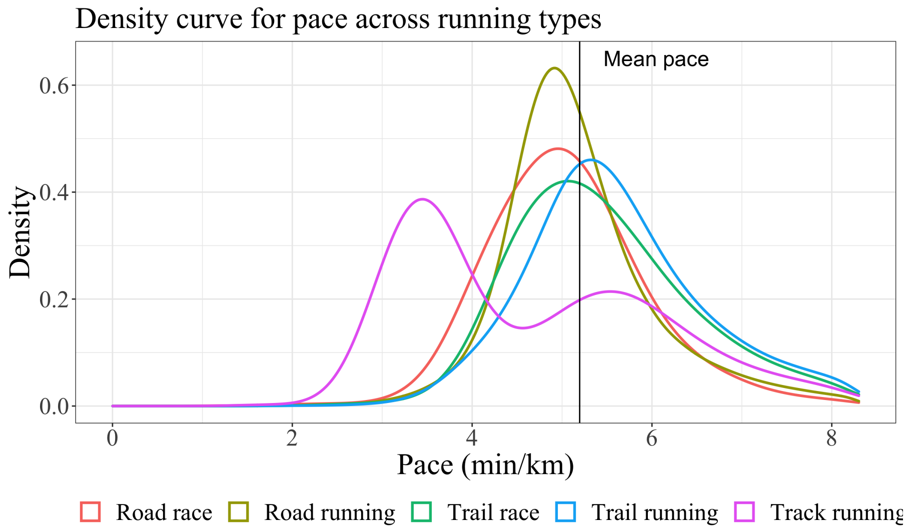


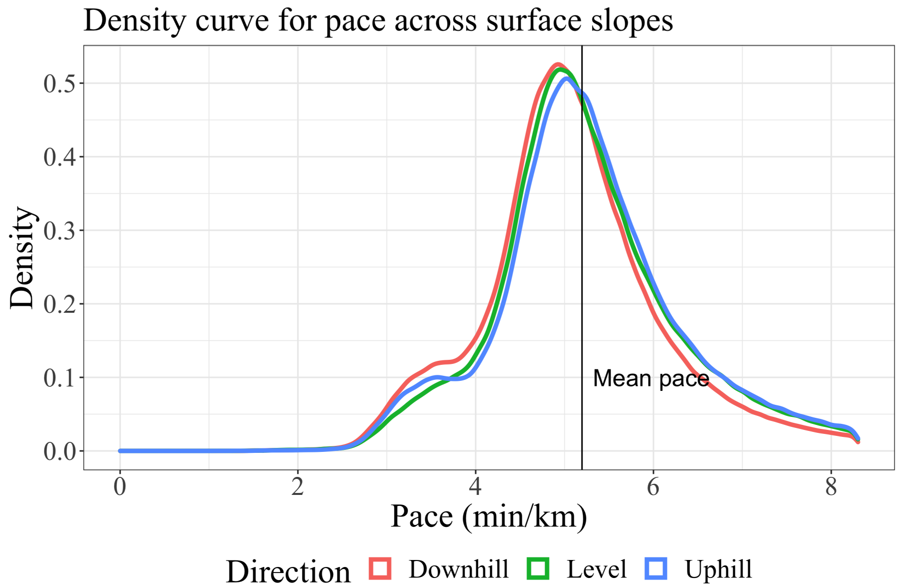
Figure 8.7: Behaviour of cadence

Performance: pace

The density curve for pace in Figure 8.8a presents with symmetrical distributions, yet the bi-modality for pace during track running is more pronounced. This is expected, as track running consists of faster and slower paced training. The overall mean pace is marked with the vertical line, at 5.2 min/km (to the right of the peaks). The density curve for pace by slope (Figure 8.8b) shows right-tailed distributions, and bi-modality for all the slopes with peaks around 3.5 min/km, preceding the major coinciding peaks. This behaviour raises some concern, since it is expected that the athlete would generally run slower going uphill than during downhill running or level running. That is, the level- and downhill running curves were expected to peak before the uphill running curve. This behaviour was also seen in Vermeulen (2018), where pace presented as almost symmetrically distributed around the 0°-slope (level ground).



(a)



(b)

Figure 8.8: Aggregate data models for pace

The runner's average pace per run is plotted over the course of nearly three years in Figure 8.9a. There is no clear indication of a seasonal pattern in the runner's performance. Generally, pace does not seem to be sensitive to the time of the day (although more dispersed for morning runs than afternoon runs), seen in Figure 8.9b, but rather to the run (or surface) type.

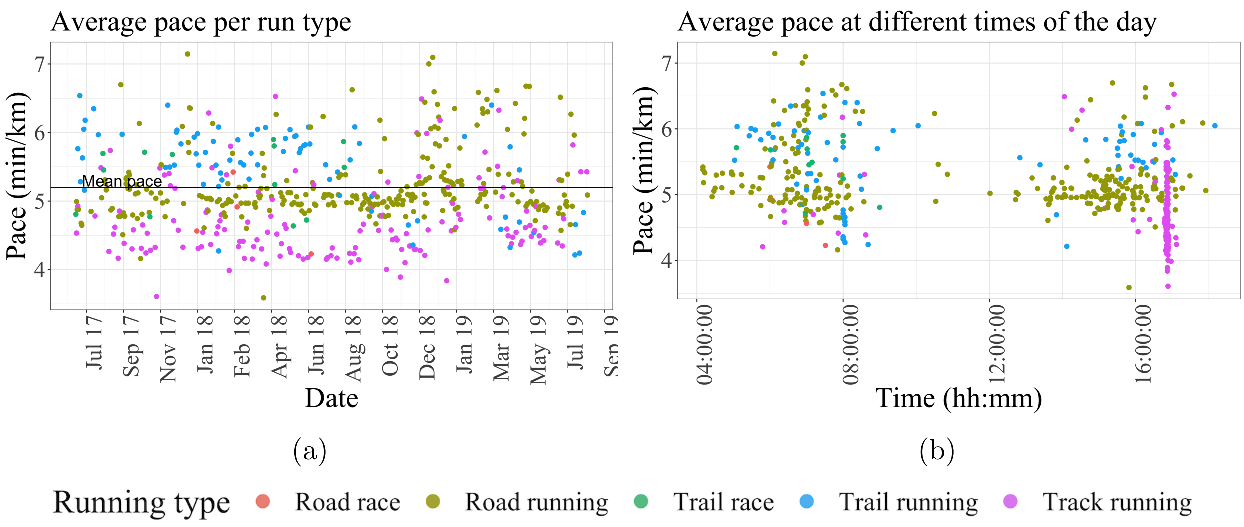


Figure 8.9: Athlete's performance over nearly three years

Recovery time

The recovery time is approximated from the interactivity time, or the time between recorded run sessions, as plotted in Figure 8.10 for intervals less than three days. Table 8.3 contains the frequency of intervals for all run sessions. The majority of runs follow within 24 hours or less of each other.

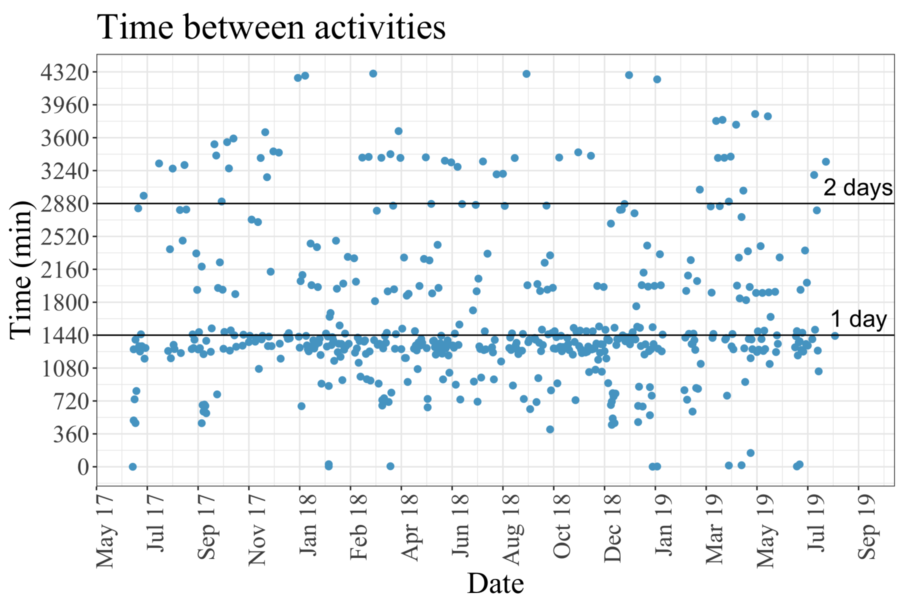


Figure 8.10: Intervals between running sessions

Table 8.3: Frequency of days between running sessions

Interval	Frequency
1 day	302
2 days	145
3 days	51
4 days	15
5 days or longer	20

Weather patterns

The overall mean temperature (± standard deviation) for the morning and afternoon time periods was 18.95°C (±6.57°C) for Pretoria over the course of one year. Figure 8.11 shows the variation in temperature per hour of the day for Pretoria.

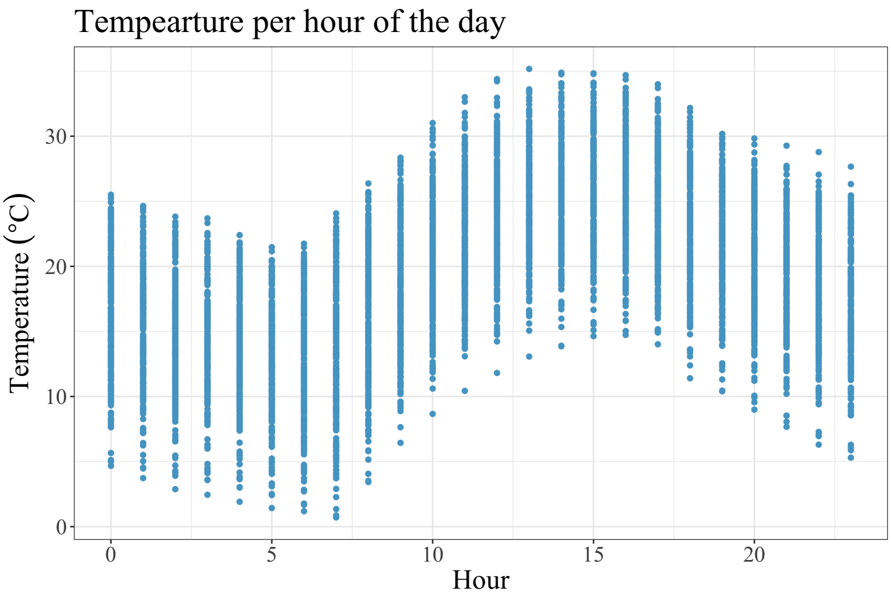


Figure 8.11: Temperature variations over 12 months for Pretoria, per hour of the day

8.3 Discretisation and complex interactions between variables

The distributions of the input parameters for the simulation model, namely cadence (transformed to steps per second, steps.s^{-1}), HR and weather patterns, were more closely studied. The input to the simulation model consisted of location specific data, namely Pretoria, which is where the athlete completed 95% of the recorded runs.

Heart rate

The HR zones and cluster analyses are based on data that have been both contextually filtered, cleaned from distribution outliers and are location-specific (Pretoria). Figure 8.12 shows the athlete's HR intensities binned into five recognised aerobic effective HR-zones from Khalil and Sornanathan (2010) at cut-off points (percentage of maximum HR): $\{65\%, 75\%, 80\%, 85\%\}$. The largest portion of HR falls within zone 2, which is within the 65% and 75% of maximum HR. The second largest portion is attributed to the highest HR zone, greater than 85% of the maximum HR. A large proportion of the time is spent close to the 180-formula's range (the aerobic zone from Maffetone (2015)) of 132 *bpm*.

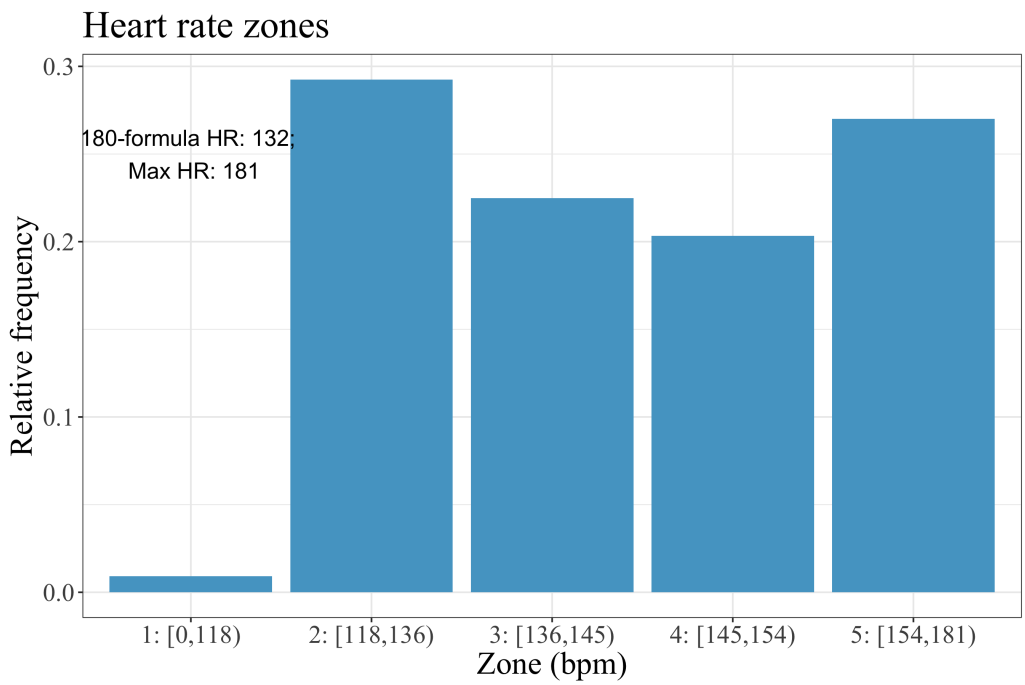


Figure 8.12: Heart rate zones and limits

The cluster analysis for HR is shown in Figure 8.13 (using eight centroids, see inset for the minimisation of the within-cluster error). The eight centroids show the central tendency of the HR behaviour towards 142 *bpm*.

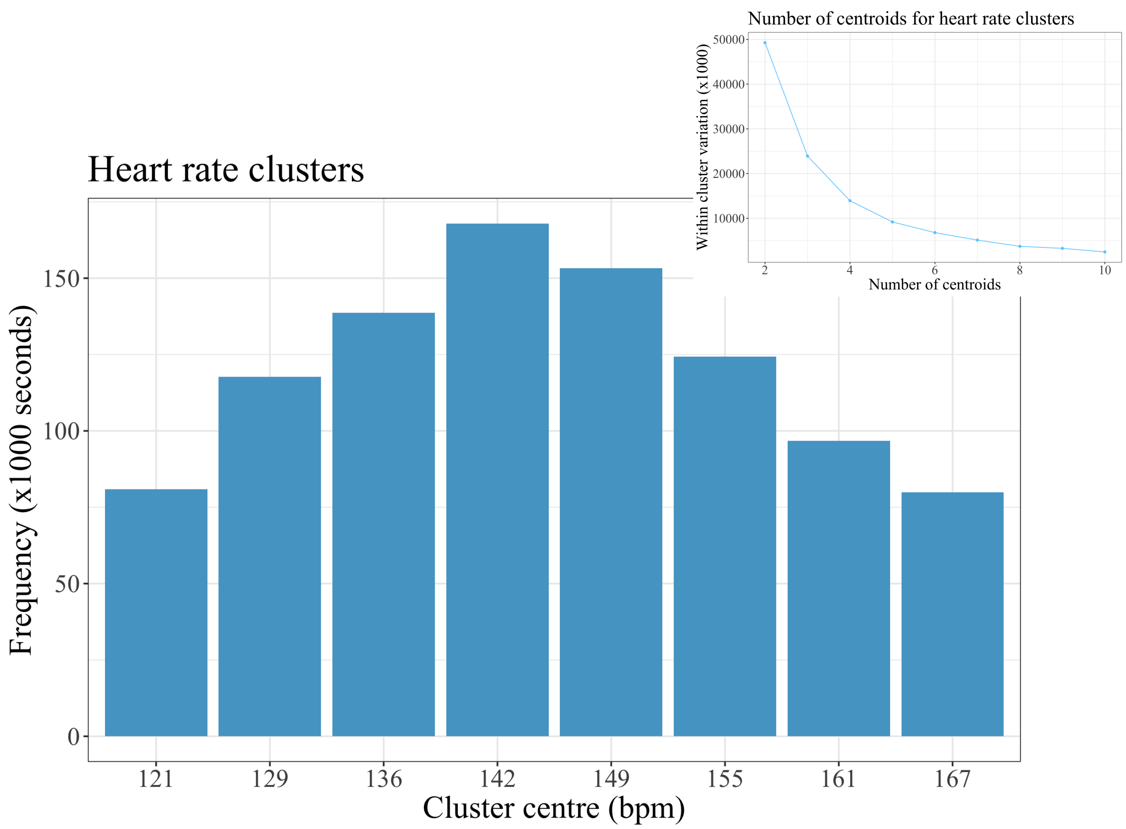


Figure 8.13: Heart rate clusters

Cadence

In Figure 8.14, the optimum number of clusters for cadence is seven, with the central tendency at 2.97 steps.s^{-1} (89 strides per minute, consistent with the cadence distribution discussed earlier).

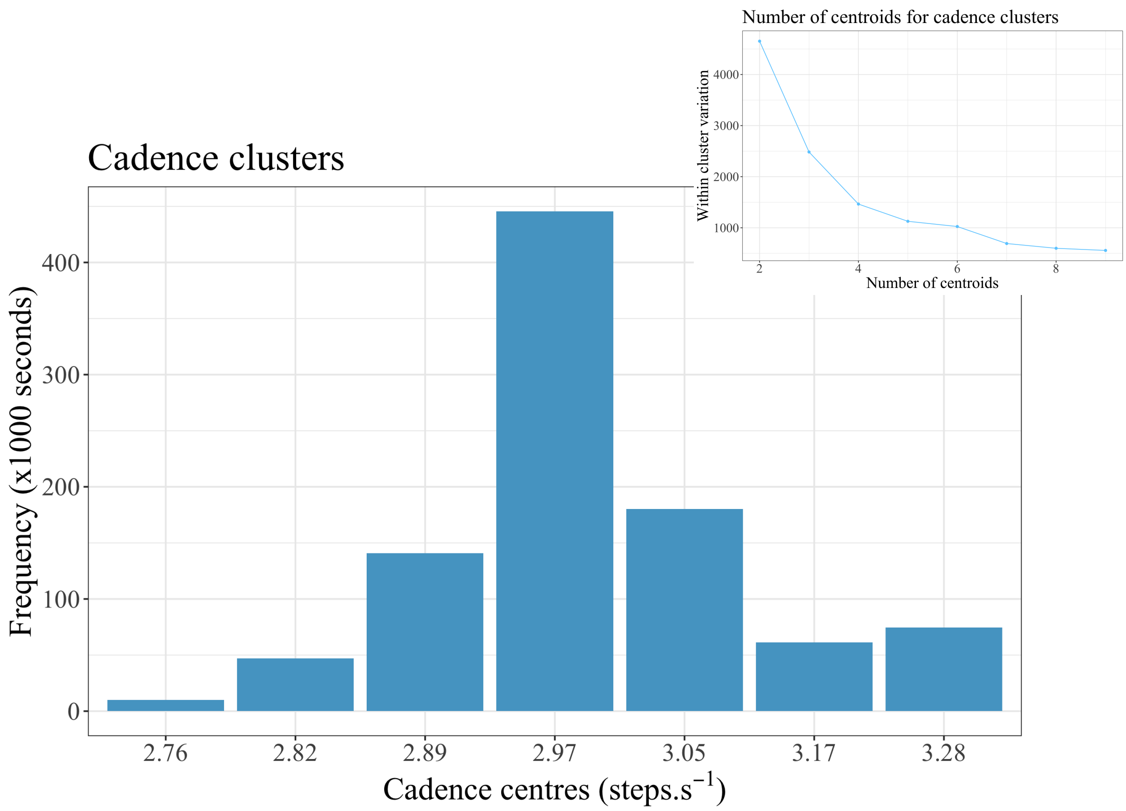


Figure 8.14: Cadence clusters

The interaction between cadence and the surface was explored by overlaying the frequency of the run type with the cadence clusters (Figure 8.15). There is dominance of cadence clusters per run type, with road running and trail running being dominated by the central cluster (2.97 steps.s^{-1}). Track running almost exclusively contains the highest cluster mean, that of 3.28 steps.s^{-1} (corresponding to 98 strides per minute).

The cluster analysis for cadence was extended for track running in Figure 8.16, to include discretisation of associated pace through the clustering of pace. Cadence was again clustered but with only two centroids, to reflect the bi-modality in the distributions seen from the histogram in Figure 8.5. Faster paces (less than 4 min/km) are associated almost exclusively with the higher cadence cluster (3.24 steps.s^{-1}, or 97 strides per minute). The structure of track running is different from road and trail running, and consists of periods of active running at faster paces for short distances followed by recovery (walking or slow jogging). It is also not reasonable to assume an athlete will be able to sustain short distance sprinting or middle distance running pace (or cadences) for the same duration as a long distance road or trail running session. This type of behaviour had to be considered for the simulation model. The largest value of pace contained in the pacing cluster center of 3.84 min/km, is 4.457 min/km. The total proportion of active track training is therefore approximated as the time spent at paces faster than this limit, which amounted to 53% of total recorded time during track running.

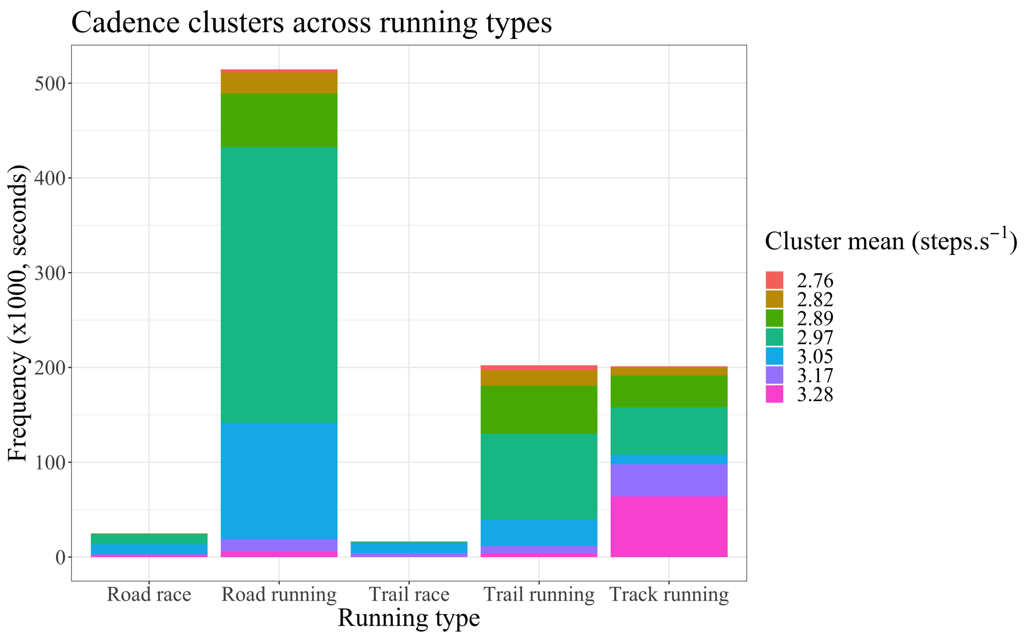


Figure 8.15: Dominating cadence clusters per run type

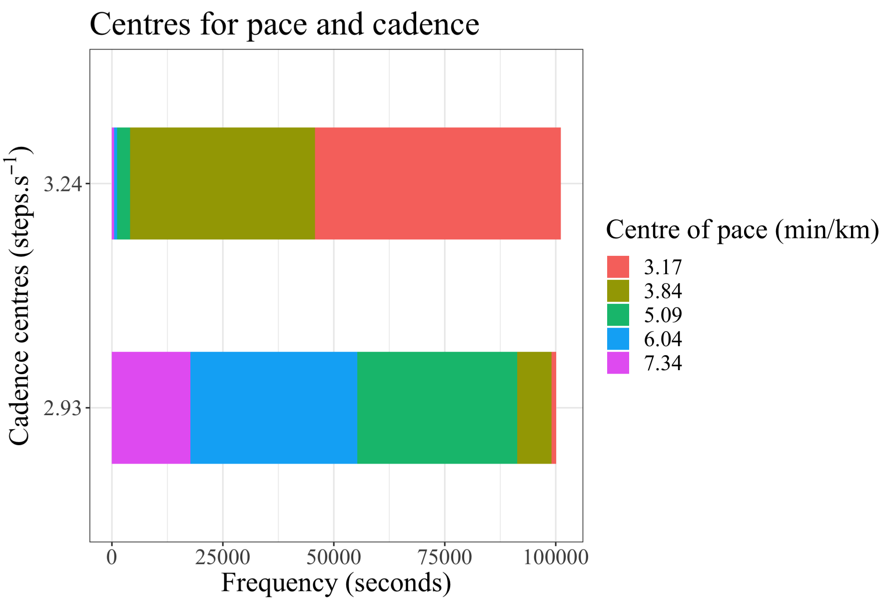


Figure 8.16: Cadence and associated pace clusters for track running

The interaction between cadence and heart rate

The analysis of the interaction between HR and cadence, briefly presented earlier in Figure 8.7d, continues here. The purpose is to find reasonable values to parameterise the interaction between the variables in the simulation model. The premise for this analysis is to develop a classification framework of cadence and HR based on levels, from which the simulation model will sample. A generic version of cadence on three levels (low, mid and high) and HR on two levels (low and high) is shown in Figure 8.17.

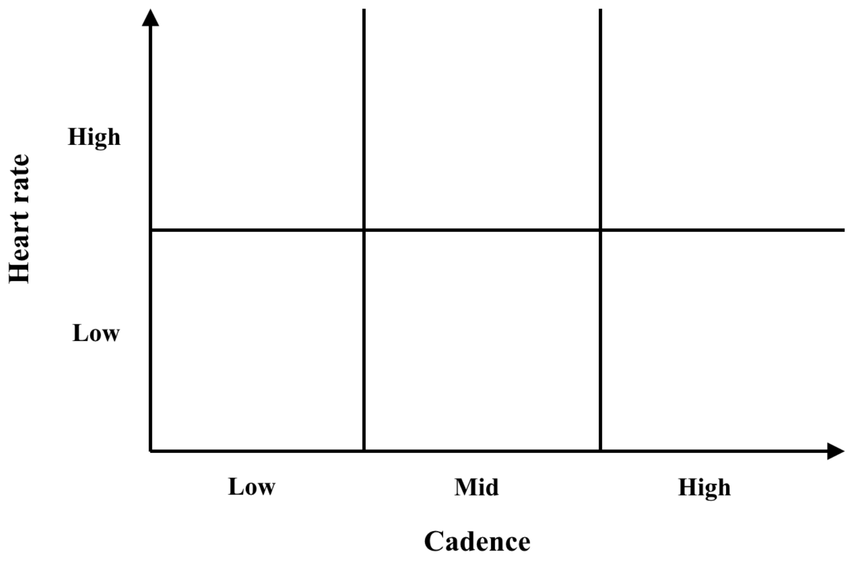


Figure 8.17: Generic levels for cadence and heart rate interaction

To create the levels, cadence was clustered with three centroids and HR with two centroids. The frequency of the cadence clusters are overlaid with the HR clusters in Figure 8.18. There is a pattern visible from the graph: although the volume (total frequency) of the cadence clusters decreases from low to high levels, the proportion of the high HR cluster (with mean of 156 *bpm*) grows across the progression of the cadence levels. That is, the high HR level becomes progressively larger in proportion of the total HR distribution per higher cadence level. This is consistent with the general upward trend from Figure 8.7d.

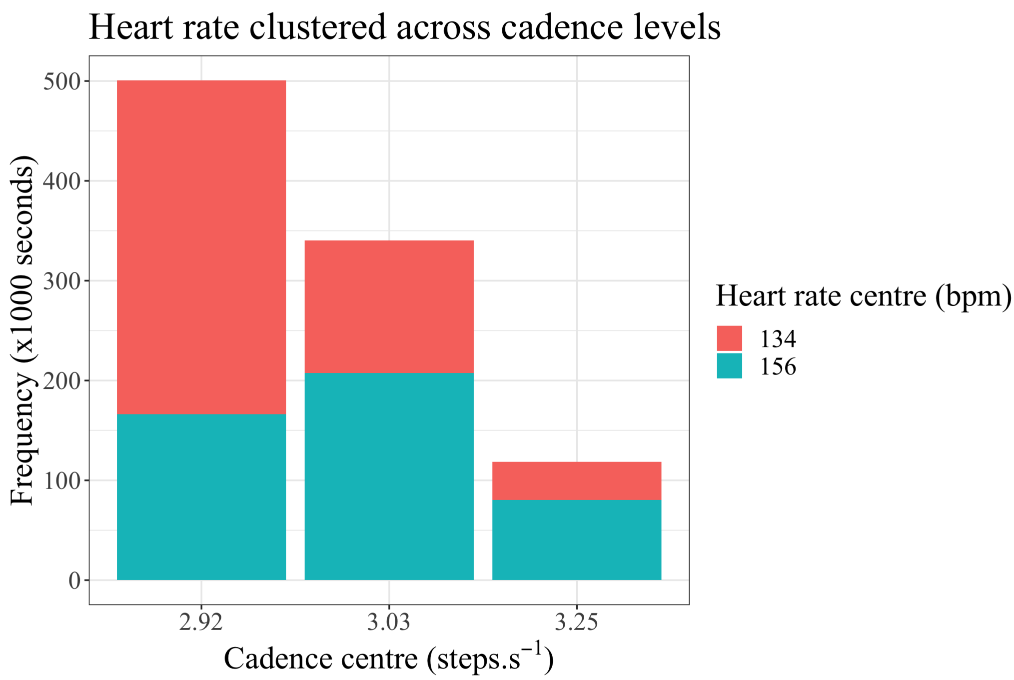


Figure 8.18: Proportional distribution of heart rate clusters per cadence level

Table8.4 provides the probability distribution of the cadence and HR clustering classification framework.

Table 8.4: Probability mass function for heart rate and cadence levels

Cadence level	**Heart rate level**	**Probability**
Low	Low	0.668
	High	0.332
Mid	Low	0.3897
	High	0.6103
High	Low	0.3216
	High	0.6784

A scatter diagram of the granular HR and cadence data points is presented in Figure 8.19, showing how cadence is demarcated by the three levels. Dotted lines indicate the centroids for HR. The solid line is the boundary between the low and high HR clusters (the minimum HR associated with the high HR cluster). This figure essentially presents the classification of the data.

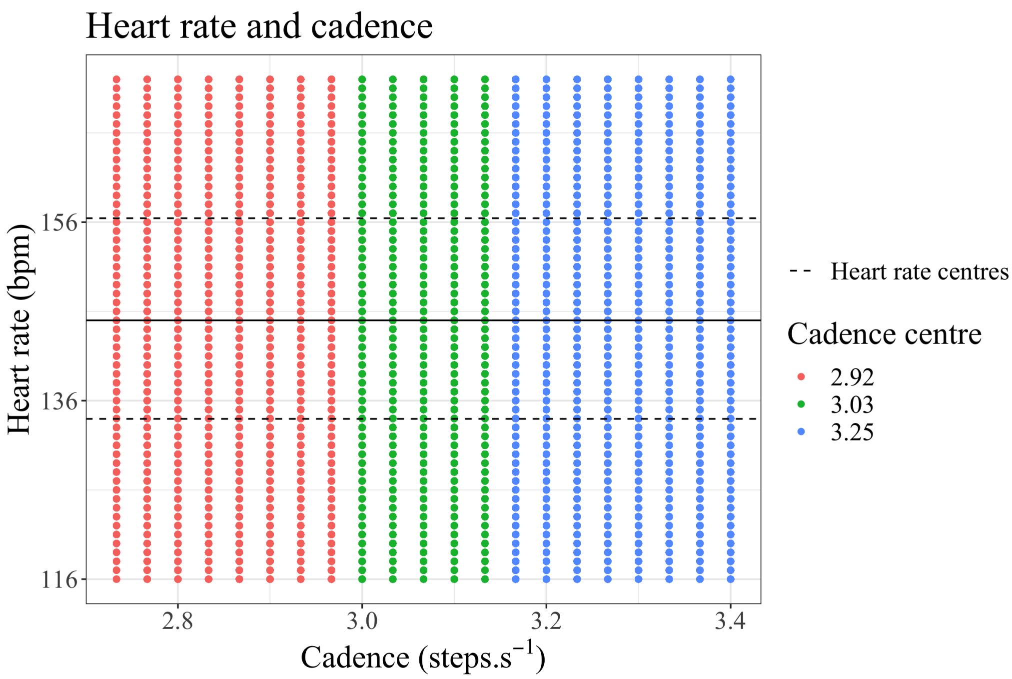


Figure 8.19: Cadence and heart rate clusters

The distribution of HR per cadence level was explored further. Heart rate for the low and mid cadence level were bell-curved and near normal, however the distribution of HR for the highest cadence level was skew to the left (Figure 8.20a). To ensure representative sampling in the simulation model, the HR distribution for the highest cadence level was transformed to be presented by the *beta* distribution, with shape parameters $\alpha = 12.39$ and $\beta = 1.7$. Heart rate was normalised by dividing all values with the maximum HR, giving a proportional value between 0 and 1. Figures 8.20a and 8.20b provide the distributions of HR for the highest cadence level and the transformed *beta* distribution. The shape parameters were estimated from the RW data, using the methods of moments (NIST, 2013; Montgomery and Runger, 2011):

$$\bar{x} = \frac{\alpha}{\alpha + \beta} \tag{8.1}$$

$$s^2 = \frac{\alpha\beta}{(\alpha + \beta)^2(\alpha + \beta + 1)} \tag{8.2}$$

Re-arranging Equations 8.1 and 8.2 provides solutions to solve for α and β respectively:

$$\alpha = \bar{x}\left[\frac{\bar{x}(1-\bar{x})}{s^2} - 1\right] \tag{8.3}$$

$$\beta = (1-\bar{x})\left[\frac{\bar{x}(1-\bar{x})}{s^2} - 1\right] \tag{8.4}$$

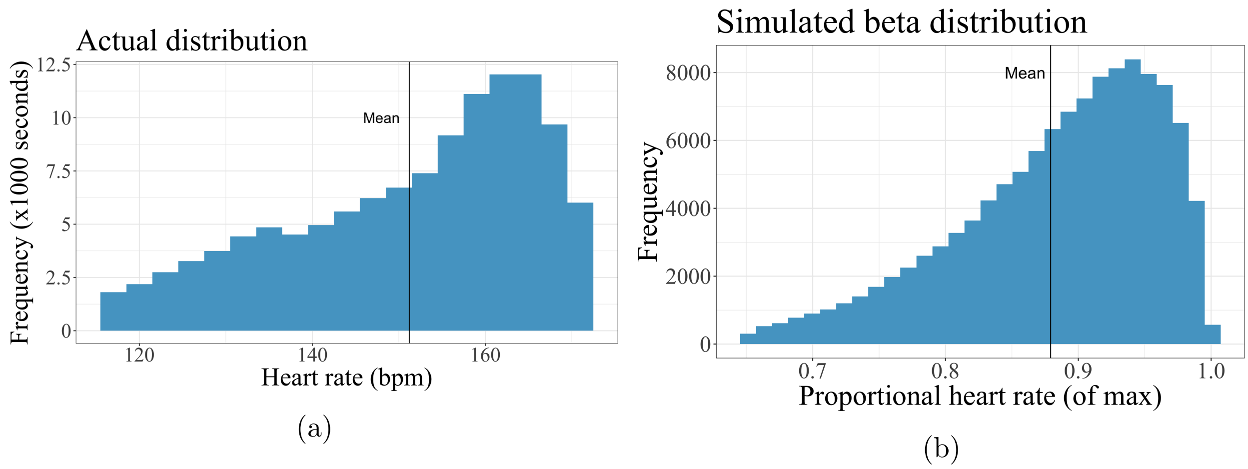


Figure 8.20: Distribution of heart rate for highest cadence cluster and the simulated beta distribution

The final classification framework is presented in Figure 8.21. The upper and lower limits between the categories are derived from the minimum and maximum values associated with the cadence and HR clusters.

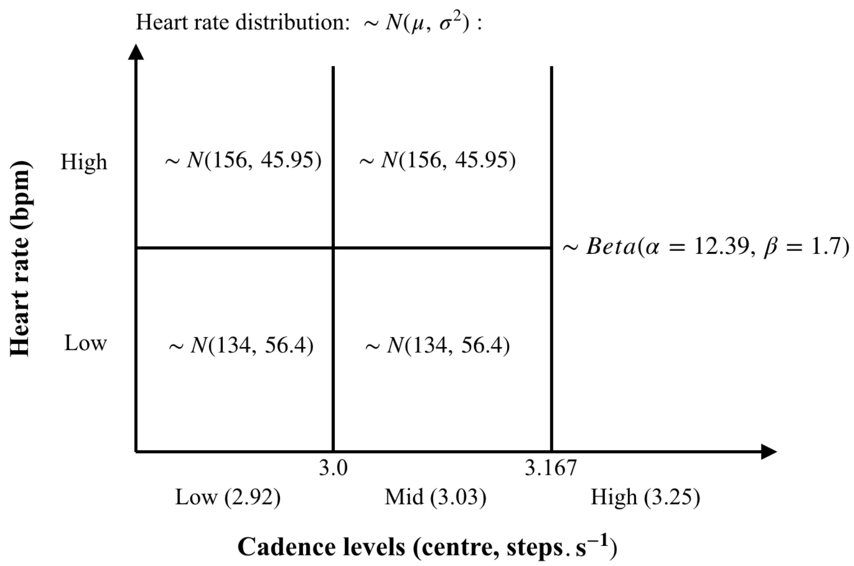


Figure 8.21: Parameters for heart rate from cadence classification

8.4 Conclusion

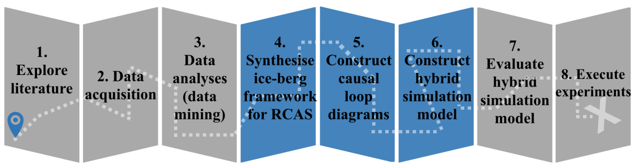
This chapter analysed the runner's digital footprint from the RW and developed the values for the sampling parameters for the simulation model in Chapter 10. The interaction between the surface type and cadence is significant and is set to influence the simulation model in the training load function. To extract the interaction between cadence and HR in a parsimonious, yet meaningful way, a classification scheme was developed to distinguish between high, mid and low levels for cadence with associated low and high levels of HR. Although subject to change based on age and resting HR, the HR zones are set to be an influential parameter in the recovery phase during the simulation of the RCAS.

Chapter 9

The generic qualitative and dynamic modelling of the athlete as a living system

Contents

This chapter formulates parts of tasks 4, 5, and 6 from the research methodology.



This chapter provides a pilot study to model the ALS using a ST approach. Qualitative models include the generic WoD and the system archetypes, followed by dynamic modelling of two archetypes in a stock-and-flow simulation model.

9.1 Qualitative models[1]

The ALS is deconstructed into a generic WoD. Sport behaviour, related to performance and injuries, is mapped to the general system archetypes.

9.1.1 The generic web of determinants

Figure 9.1 is a generic version of the WoD for an ALS. The WoD is subset into the internal and external elements from Brukner and Khan (2006), showing only interactions and not the direction of influence. Biomechanics is a grouped term for muscles, joints and the skeletal structure, since these function as a mutually inclusive unit: each muscle is connected to a bone

[1]parts of this section were published in *Theoretical Issues in Ergonomics Science*, July 2020

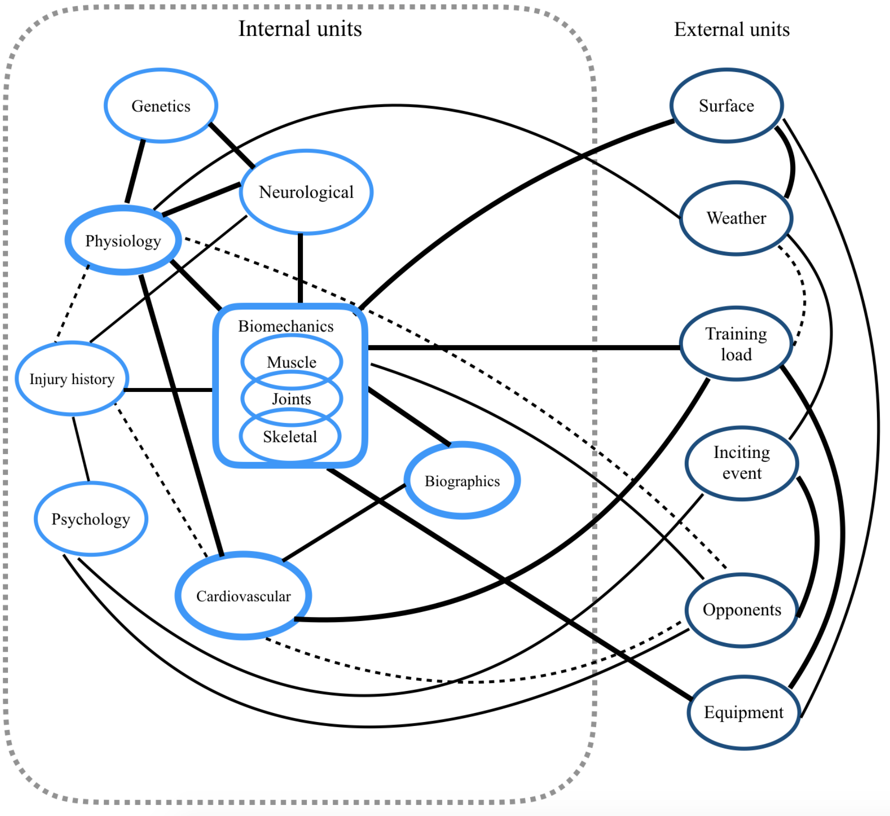


Figure 9.1: The generic web of determinants for an athlete

in the body and each muscle contraction exerts a force on a bone. The risk factor classifications from § 4.3 *Risk and protective factors in running* are used in the web, with some modifications. The boundary for the internal elements of the ALS is the athlete's skin: consider what the athlete will take with them when they fall asleep. Therefore injury history is considered as an internal element, since the scars (or milestones) from an injury reside within the athlete's body. Training load is rather an external, modifiable factor than an intrinsic property. The same *modus operandi* in Bittencourt et al. (2016) is followed to draw the web:

- Thick lined circles have more interactions than circles with lighter lines, subsequently they exert a stronger influence on the outcome.
- Thicker, solid lines represent stronger interactions, while dotted lines are weak interactions.

Interaction amongst the elements may be internal with internal, internal with external and external with external. For instance, there exists interaction between the weather and the surface: roads become more slippery (or muddy) in wet weather; between surface type and equipment: a runner would prefer trail running shoes instead of road running shoes when running on dirt or single tracks; between equipment and training load: shoes will wear out more due to higher frictional forces on tough, hard surfaces than softer ones.

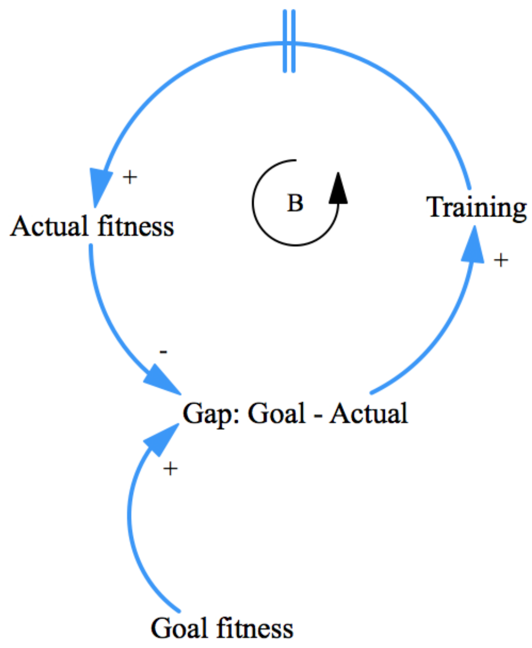
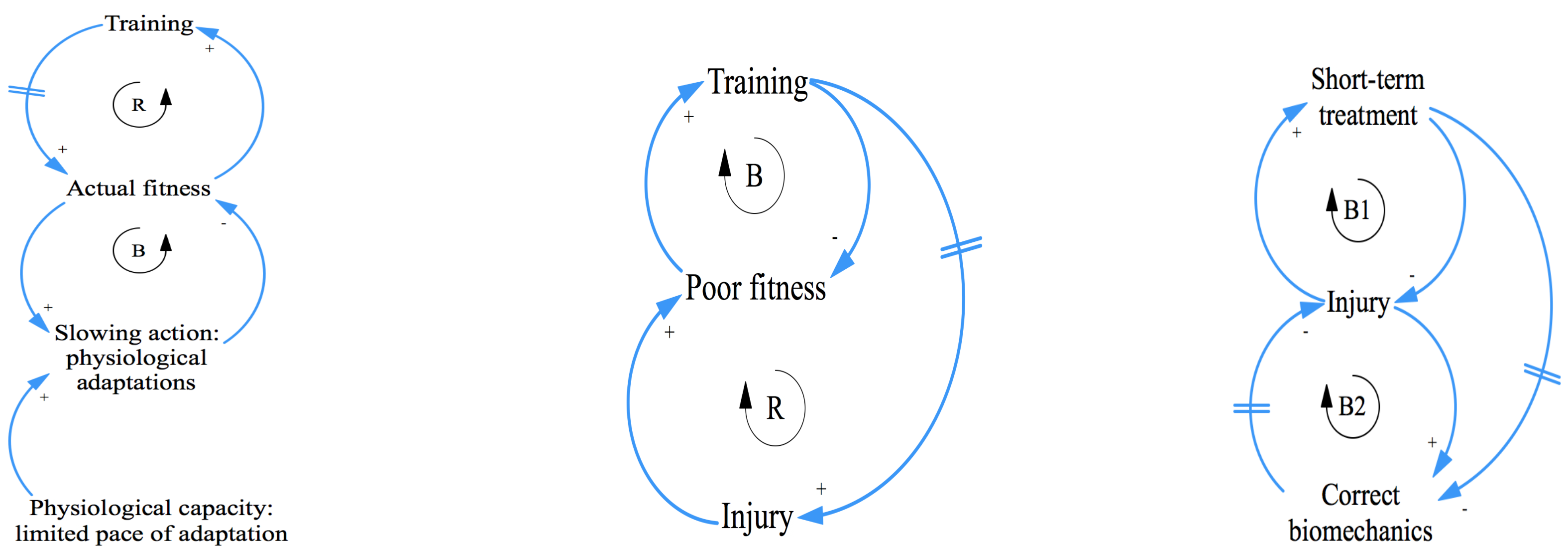


Figure 9.2: Fitness as a gap driven balancing loop

9.1.2 The basic system archetypes in sport

The training cycle is simply a goal-driven balancing loop, seen in Figure 9.2, from which the archetype systems originate. In the training cycle, there is a gap between an athlete's actual fitness levels and their goal fitness level. This gap drives the training initiative. Training takes time (a delay) to build (add to) actual fitness. When fitness is added, it closes the gap between the goal and the actual fitness. As the gap closes, training stabilises. Fitness may also be thought of the amount of structure in place to accept the physical load imposed by training. The six basic system archetypes (Figures 9.3 and 9.4) were identified (refer to § 6.3.3 *System archetypes*) that are eloquent, high-level representations of the ALS in the training cycle.

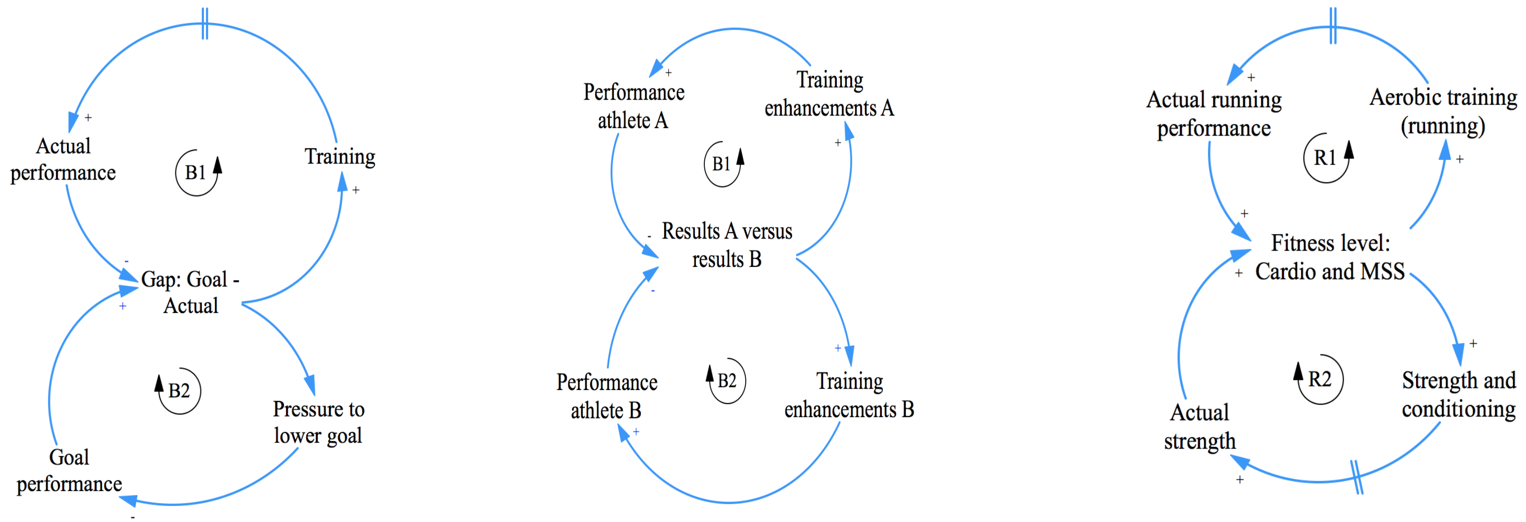


(a) Limits-to-growth: pace of physiological adaptation. An athlete starts with training to increase fitness; however, there is a delay between the training and fitness benefits. Actual fitness requires physiological modulations of the athlete's bodily structures, which might occur at a slower rate than expected. The athlete's body has a physiological capacity to adapt at a certain pace. This delay in adaptations limits the growth of their fitness levels.

(b) Fixes-that-fail: too much or wrong training. Poor fitness of an athlete would instigate increased training loads, either in diversity, intensity, volume or a combination. However, an unintended consequence of sudden increases and uncontrolled training are overuse injuries (Herring and Nilson, 1987; Larsen et al., 2016). Should an insidious injury go unchecked, or are simply ignored by the athlete, this unintended consequence exacerbates the already poor performance. In this case, more training (the 'fix') is actually causing the problem it intended to solve, that is, the 'fix' had failed.

(c) Shifting-the-burden: a 'quick fix' to solve a debilitating symptom of a deeper, underlying problem. The burden is shifted to over-reliance on short-term treatment, instead of attention paid to the fundamental solution. Short-term treatments include release of muscle spasms, pain medication, and joint bracing aimed at returning the athlete to participation despite the body not being in an optimal fit state. The more athletes rely on quick fixes with immediate results, the more their body maladapts with long-term injuries (often colloquially coined 'niggles' by commentators) as an almost permanent outcome.

Figure 9.3: Basic system archetypes: limits to growth, fix-that-fail, shifting-the-burden



(a) Drifting goals: setting realistic expectations. In the athlete's case, this archetype might rather be termed as 'altering goals'. The rationale is for athletes to find the realistic goals for their capabilities, and appropriate for their age. A set goal and the actual performance creates a gap, which drives progressive training by the athlete to close the gap with passing time. However, should this goal become unattainable, there is pressure on the athlete to lower the goal to an achievable level. In contrast to the progressive training, the lowered goal instantly closes the gap, allowing the athlete to perform satisfactorily for their age and capabilities.

(b) Escalation: uncontrolled growth or decline. Results from competing runners are evaluated against each other, and athlete A is performing better than athlete B. Athlete B increases their training efforts or make use of other performance enhancements to gain the edge over their competitor. Athlete A retaliates by increasing their own effort when the athlete B's results threaten their position. This vehement behaviour from both parties drives the perpetual pursuit of methods to enhance performance, leading to faster running times, breaking records and pushing the boundaries of human performance.

(c) Success-to-the-successful: unbalanced training. One subsystem of the ALS is strengthened and another neglected. In the case for the runner, high cardiovascular fitness is pursued at the cost of mechanical strength of the MSS. Strengthening the MSS is neglected because the integrity thereof is not an obvious requirement for endurance sport. As more resources (specifically the runner's time) is devoted to running itself, less is spent on strengthening and conditioning through exercise therapy. Eventually the MSS fails since it has not developed the capacity to handle the cyclical overload.

Figure 9.4: Basic system archetypes: drifting goals, escalation, success-to-the-successful

9.1.3 More complex system archetypes

Two complex system archetypes, growth-and-underinvestment and tragedy-of-the-commons, are made up of different combinations of the basic archetypes. Growth-and-underinvestment is a combination of limits-to-growth, shifting-the-burden and drifting goals (Figure 9.5). Tragedy-of-the-commons combines reinforcing loops with limits-to-growth systems (Figure 9.6).

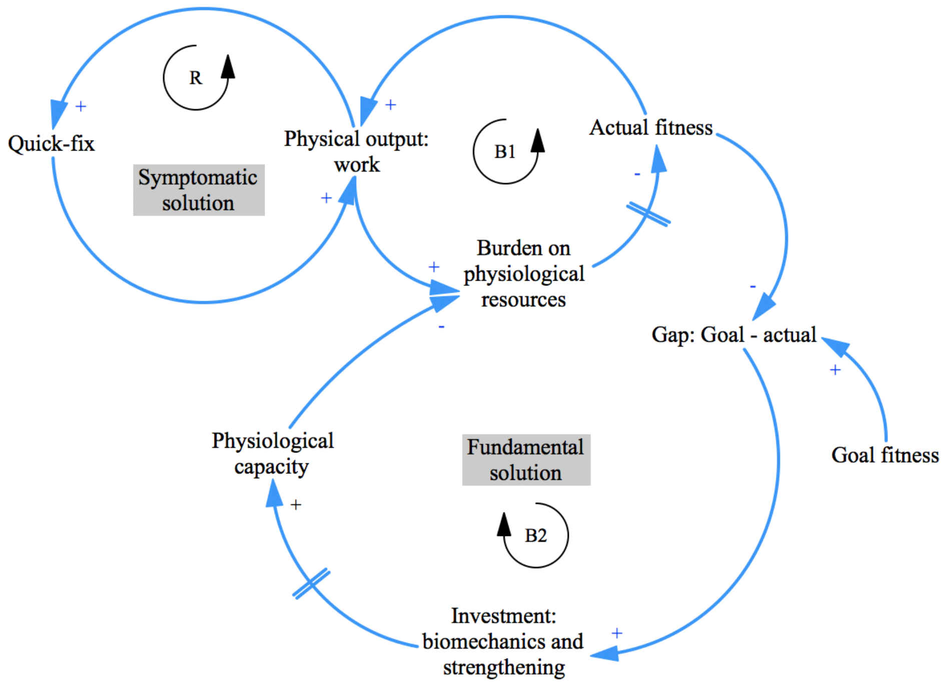


Figure 9.5: Growth-and-underinvestment: quick fixes and fundamental solutions. By pushing on the reinforcement loop (more of the same training) and not investing time and effort in gait retraining, biomechanical adaptations through strength and conditioning or recovery techniques, will eventually reverse the fitness levels into a downward spiral. Athletes who do not invest time and/or resources in recovery and fruitful training to enhance fitness *and* health, yet continue in their set training plans and utilise aids such as extra bracing, special insoles, pain medication and massaging to 'soothe' the symptoms. Athletes neglect addressing the underlying issues due to faulty biomechanics and poor recovery. In this way, athletes opt for symptomatic solutions instead of fundamental ones, putting these off into the future and perhaps negating their body's ability to heal itself. This figure was published in *Theoretical Issues in Ergonomics Science*, July 2020.

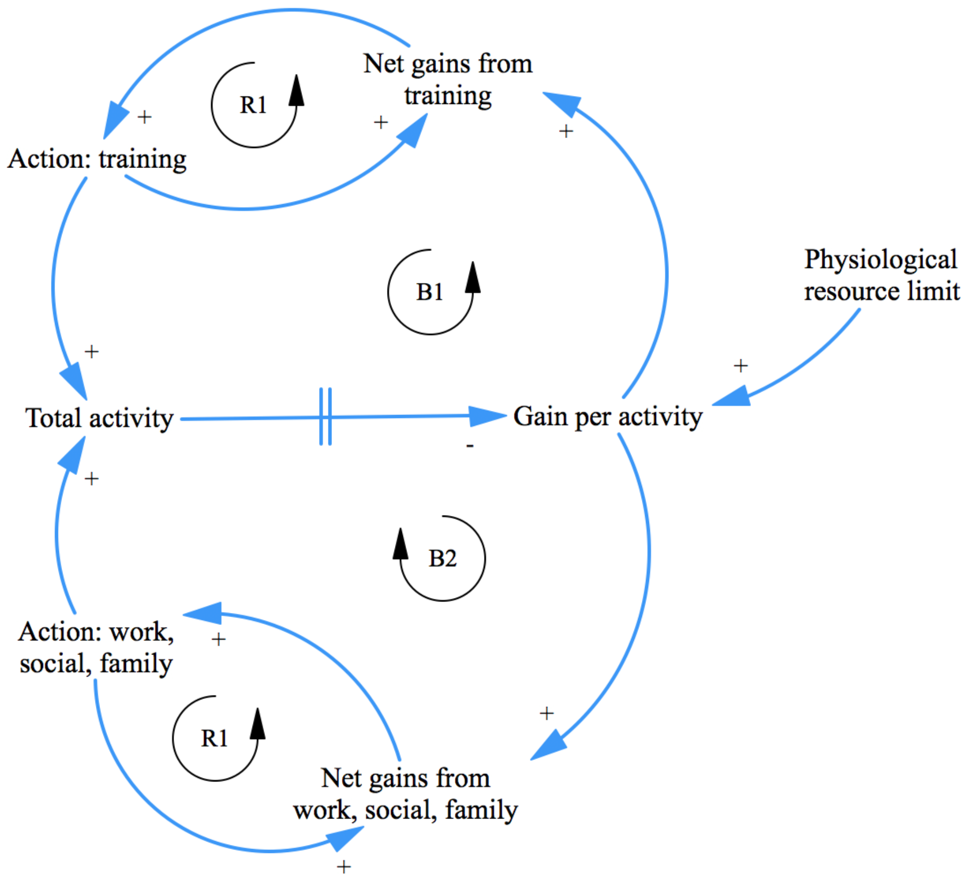


Figure 9.6: Tragedy-of-the-commons: exploitation of a common resource. The athlete's body can be viewed as the shared resource from which all life's activities draws energy: work, family, social activities and training. Neglecting recovery to restore structures of the body will deplete the resources available and the ALS becomes an unsustainable system. The clinical representation of a depleted system is the OTS, discussed earlier in 4.6 *The overtraining syndrome*.

9.2 Pressure to improve performance as a growth and underinvestment system[2]

The growth-and-underinvestment archetype (Figure 9.7) incorporates the debate in innovative shoe design featuring the polarising Nike Vaporfly and Alphafly road running shoes (discussed earlier in § 4.4 *Domain specific solutions towards injury prevention and performance improvements*). In the growth-and-underinvestment archetype, the running shoe represents the quick fix, growing action that delivers instant results to gain running speed and decrease energy consumption; illustrated in the top-left reinforcing loop (R) as the symptomatic solution to perceived under-performance (slower pace). Whilst instant results (faster paces) are achieved when running in the shoes, the strain on the body's physical resources continue, whereby the performance output still places a burden on the body's structural and physiological resources to adapt to the constant loading. In the case of uncontrolled growing actions (over-reliance on shoes and eventual replacement of worn-out shoes that are no longer effective, similar to the aggressive marketing campaigns in the industrial example earlier), start to negatively influence the actual fitness and subsequently lowers their output or limits the growth thereof (the top-right balancing loop, B1). Simultaneously a gap opens up between the athlete's actual fitness

[2]this section was published in *Theoretical Issues in Ergonomics Science*, July 2020

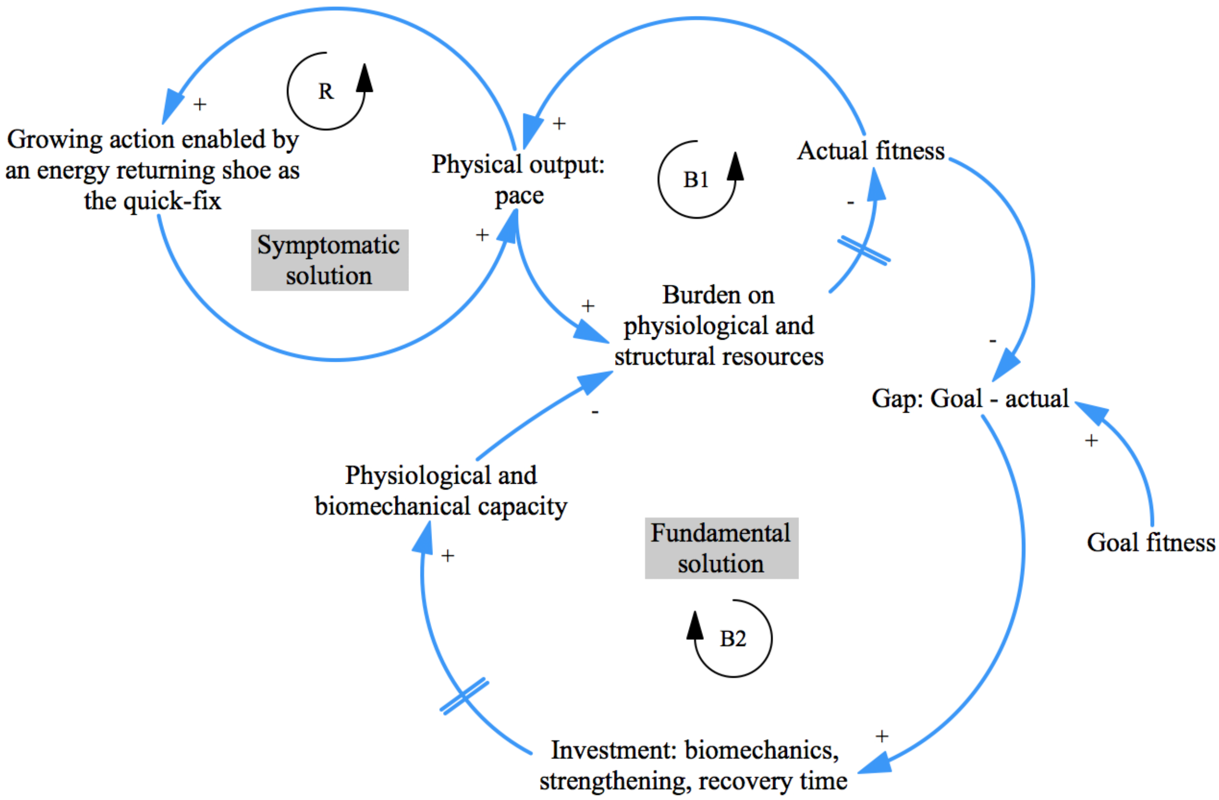


Figure 9.7: Shoes as a quick fix in the growth-and-underinvestment system. This figure was published in *Theoretical Issues in Ergonomics Science*, July 2020.

and the goal fitness required for desired performance. This gap drives the need for investment in the fundamental solution: to build structural biomechanical and physiological capacity that will lessen the burden on the body's structures for the athlete to become resilient and healthy (the bottom balancing loop, B2). Capacity building includes gait retraining (Barton et al., 2016), aerobic base build-up, mindful recovery (Maffetone and Laursen, 2016; Kreher and Schwartz, 2012; Meeusen et al., 2013) and other longer-term solutions.

As with the industrial example from Figure 6.6, each iteration of the symptomatic solution with near instant results ('it worked well the previous time', 'why change something that works?'), the fundamental solution is put off further into the future. The added cushioning and elastic return from the shoe's midsole material lowers energy demands from the runner (Burns and Tam, 2019), taking away the need to invest in biomechanical training and conditioning to improve the leg stiffness and other gait mechanisms to augment the innate characteristics of the athlete that enhance performance and protect against injury. The additive, overall burden on the resources of the runner becomes more sever over time; eventually this burden reverses the output through negated fitness. The athlete is either at a higher risk for injury, perhaps already injured or in an overtrained state.

Other growing actions or symptomatic solutions to decreased fitness and lower output may be even more training (from the 'no-pain-no-gain' mentality (Maffetone and Laursen, 2016)), compression wear to deal with circulatory shortcomings, orthrotics that instantly correct malalignments, taping techniques to enhance proprioception and so forth (Barton et al., 2016). By not allowing time for active recovery, gait retraining and strengthening, that is, by disregarding the delay between investment and capacity, the body simply cannot follow the fundamental loop to rebuild structure when its neurological systems, the MSS and the supportive CVS are in disarray brought on by the accumulated burden on resources. Coaches and athletes may use the growth-and-underinvestment archetype in an iterative manner to differentiate between short- and long-term solutions, in order to draft a flexible training program that will achieve performance goals and protect against injury.

9.3 Dynamic modelling of the archetype systems[3]

The stock-and-flow diagram for the generic training cycle is shown in Figure 9.8. The objective is to close the gap between the athlete's current fitness and the desired level. The model is an extension of the basic balancing loop between training load and structure.

In the simulation, fitness is captured as the level of structure in an athlete's body, concerning two of the subsystems that make up the ALS, namely: cardiovascular fitness and musculoskeletal strength. Training load is modelled as a pulsed input every two days and is a function of the power output by the athlete. Each training session inflicts some micro-damage on the body (§ 3.3 *Training: a function of biomechanics, physiology and the neuromuscular pathways*), which is repaired within a certain time frame. Each fibre that is micro-damaged also initiates the body's response to synthesise new fibres, that is, the muscles are both growing, vasculature is improved and the athlete's structure (fitness) is enhanced. Each structure increases the strength (force output) from the athlete's body, positively influencing the power output and subsequently the training rate may be adapted. This interaction between micro-damage, structure levels, breakdown and training load must not go unchecked. The path to an unintended consequence, injury, is modelled as the additive damage the body incurs, both as a normal result from micro-damage, but also due to inadequate recovery. This behaviour is modelled using the ratio between the damaged structures and the healthy structures. Once a certain threshold in the ratio is reached, the body's repair delay increases, since the body cannot maintain structure repair and formation in the presence of uncontrolled damage to tissue. Over time, the training load compounds this effect, until the damage:structure ratio exceeds a manageable range and tissue cell death (tissue failure) occurs rapidly. This event represents a catastrophic injury, such as a stress fracture or tendon rupture.

[3]this section appears in proceedings for the *7th Annual System Dynamics Conference*, System Dynamics Society South African Chapter, November 2019

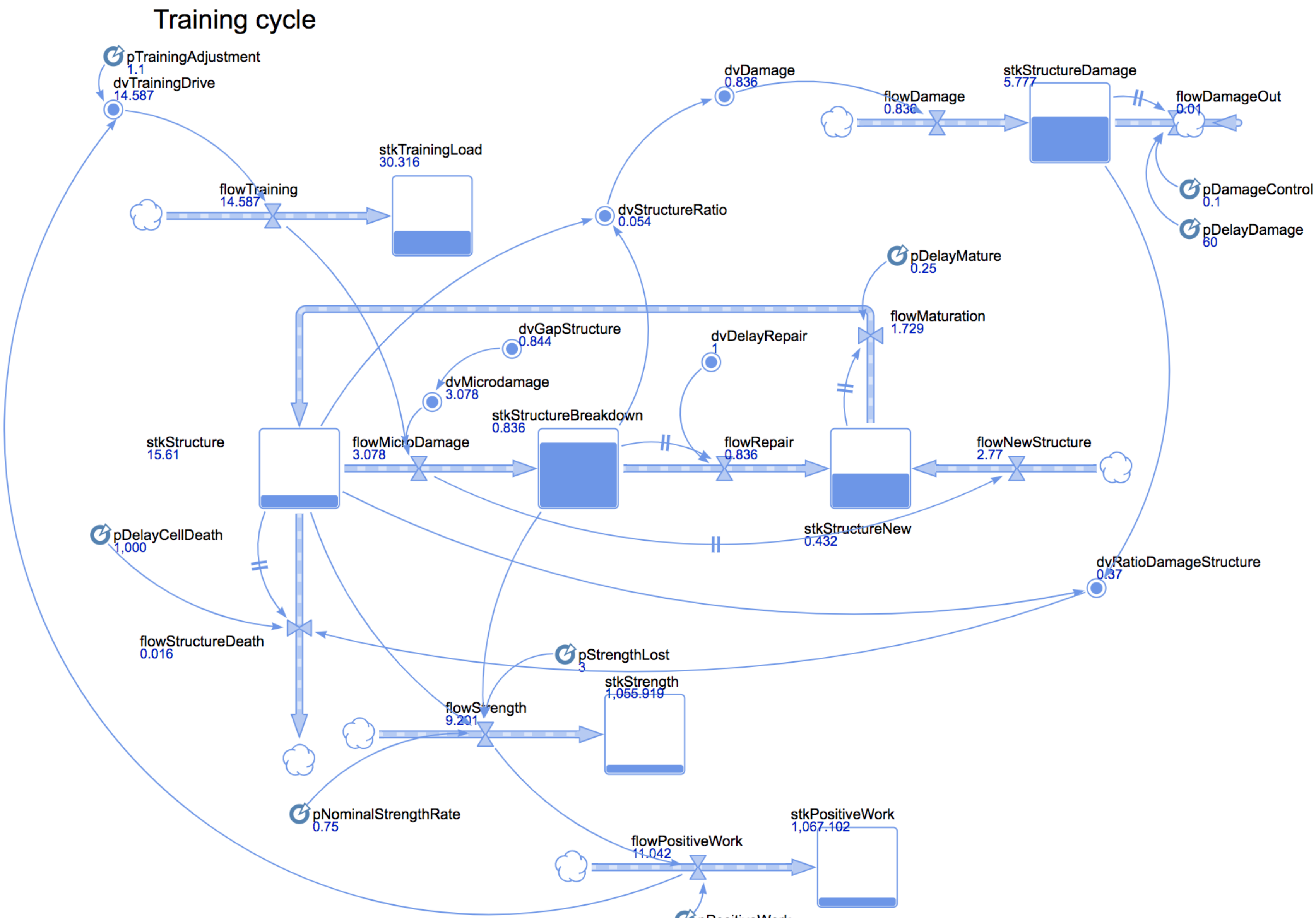


Figure 9.8: Stock-and-flow structure for the training cycle. This figure appears in proceedings for the *7th Annual System Dynamics Conference*, System Dynamics Society South African Chapter, November 2019

9.4 Dynamic behaviour of the archetype training cycles and injuries[4]

The results from two athlete archetypes' behaviour simulated with the generic stock-and-flow model over time are shown in Figure 9.9 (fix-that-fail) and Figure 9.10 (shifting the burden).

In the fix-that-failed system, the athlete did not release the additive damage (through proper recovery, alternative training modalities and other mechanisms), yet continued to increase the training load. There is an inflection point just beyond 100 days, the result of damaged tissue but not above the catastrophic failure threshold. The athlete's training load, strength and power output dropped momentarily. The athlete responded to this decrease in fitness by continuing training (albeit with a longer tissue repair delay, negatively impacting the rate of power production). Although fitness (structure formation) improved over time and the athlete came close to their goal with over 90% of the goal reached, a catastrophic tissue failure occurred after day 220.

In the shifting-the-burden system, the athlete did take care of the additive damage, but not sufficiently by ignoring the fundamental solution (to fix their biomechanics) and opted instead for quick solutions with short-term effects. The result is that the goal attainment percentage stabilised close to 100%, yet an injury was inevitable. The injury did come later than the fix-that-failed system, nonetheless the effects of the additive damage was severe enough to incur a serious injury which will side line the athlete for considerable time.

9.5 Conclusion

Typical sports behaviour was mapped to the system archetypes, of which two were then dynamically modelled in a stock-and-flow simulation model. The generic modelling of archetypes served as an illustration of steps 2 and 3 from the STMM (§ 6.2). It is now time to proceed to model a specific sports case, that of the RCAS in Chapter 10.

[4]this section appears in proceedings for the *7th Annual System Dynamics Conference*, System Dynamics Society South African Chapter, November 2019

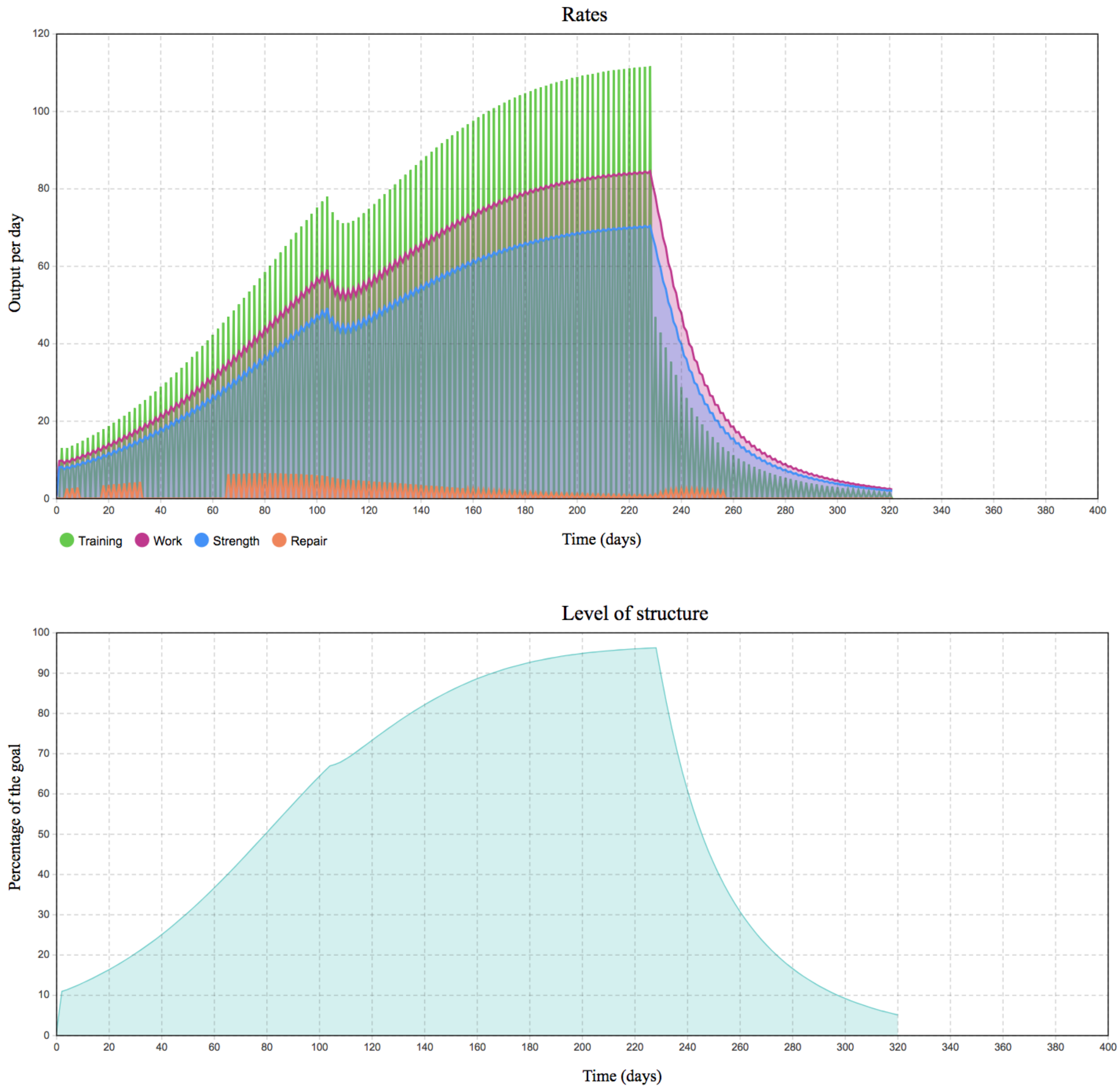


Figure 9.9: Dynamic behaviour of the fix-that-failed system. This figure appears in proceedings for the *7th Annual System Dynamics Conference*, System Dynamics Society South African Chapter, November 2019.

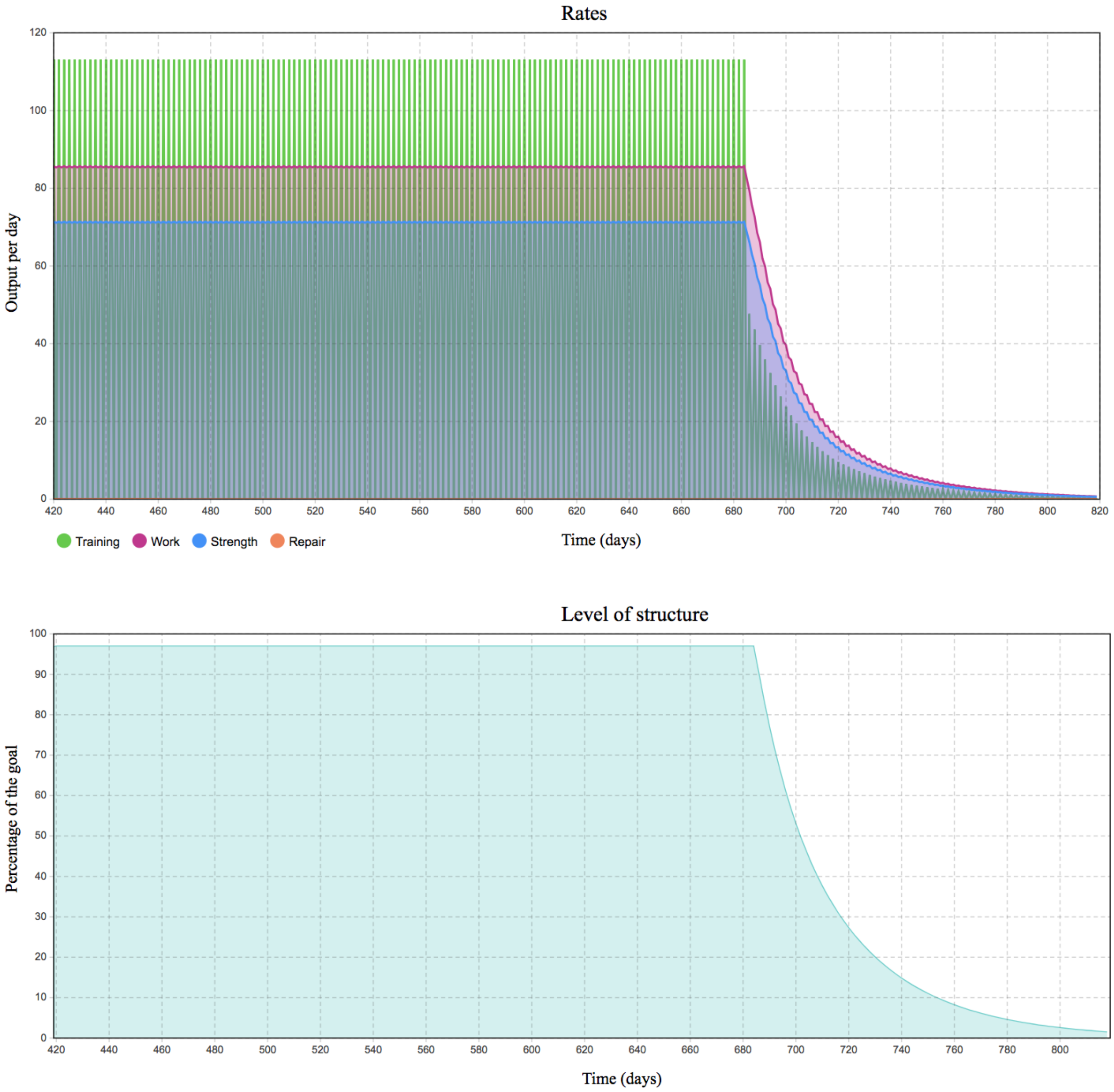


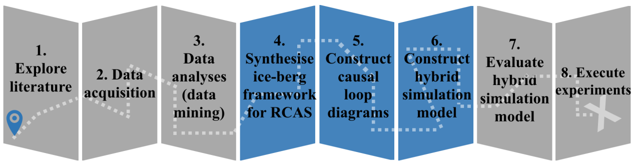
Figure 9.10: Dynamic behaviour of the shifting the burden system. This figure appears in proceedings for the *7th Annual System Dynamics Conference*, System Dynamics Society South African Chapter, November 2019.

Chapter 10

Modelling the runner as a complex, yet adaptive, system

Contents

This chapter formulates tasks 4, 5, and 6 from the research methodology.



The RCAS is a special case of the generic ALS from Chapter 3. The qualitative and quantitative modelling methods of the ST approach are applied to the RCAS. Qualitatively, the RCAS is decomposed by means of its WoD, and its emerging behaviour is shown through the ice-berg model (§ 10.1) followed by the CLD, namely the rCLD (§ 10.2).

The theory, pilot models, and concepts developed in earlier chapters culminate in the quantitative hybrid simulation model, and is presented in § 10.3 (see Figure 10.1). The simulation is parameterised with primary, secondary and illustrative data. The primary data (from the RW and the weather data bank, *MeteoBlue*) were analysed earlier in Chapter 8. The physics concepts applied (to model the runner as a spring-mass system and the process of entropy) were developed in Chapter 5. The results from applied interventions follow in Chapter 12.

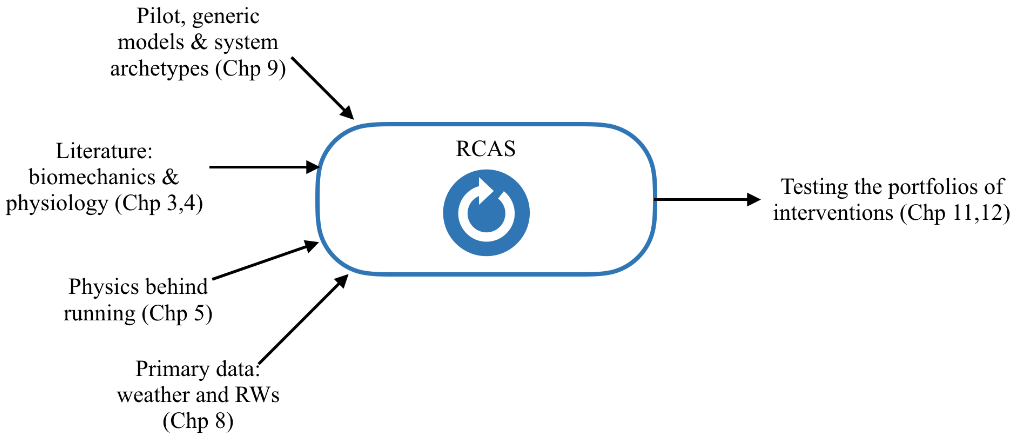


Figure 10.1: Theory and data contributions to the simulation of the RCAS

10.1 Describing emergent running performance from interactions using the ice-berg model[1]

The ice-berg framework is used to facilitate the systemic thinking explained by Bartlett (2001): dissecting the complex system of running into its parts and synthesising the system's behaviour through their interactions. The thought experiment presented here is to first analyse the runner as a system, by breaking it down into manageable components at different levels of visibility, and then string these together again through a step-by-step synthesis into the expected behaviour as a whole. In this way, avenues of behaviour are explored as subcomponents generating the behaviour of the whole.

Figure 10.2 presents the WoD of the RCAS, distilled into the ice-berg framework from Figure 6.3. The WoD is subset into two groups: dynamic and structural. The interaction between the structural elements give rise to the elements in the dynamic web, and the interaction between the dynamic elements alter the interactions between the structural elements. Interaction lines are left out of the WoD as not to clutter the visualisation. Running is an activity in which a multitude of interactions takes place in a multiplanar dimension involving the entire body, thereby creating a rather extensive web. Physical output is in terms of work done by the MSS (to move the body overground), the CVS (circulating the required fuel, oxygen and removal of waste products) and their combined effect that give rise to either an injury, or a healthy body. The two events are mutually exclusive: an athlete is either injured or healthy (complete absence of any injury). Feedback originates from the events and physical outputs and should travel back to the WoD and the training philosophy.

The training philosophy reflects the mental models of athletes, coaches and practitioners: it necessitates perception of the structural make-up; that is, how the athlete *thinks* their body is structured. Applying the training philosophy reflects how physiological and biomechanical governing laws of the ALS are leveraged to condition the athlete for optimal performance, for instance strength training to improve muscularity, flexibility to relieve stiff joints and HR based aerobic training to augment heart function.

[1]parts of this section were published in *Theoretical Issues in Ergonomics Science*, July 2020

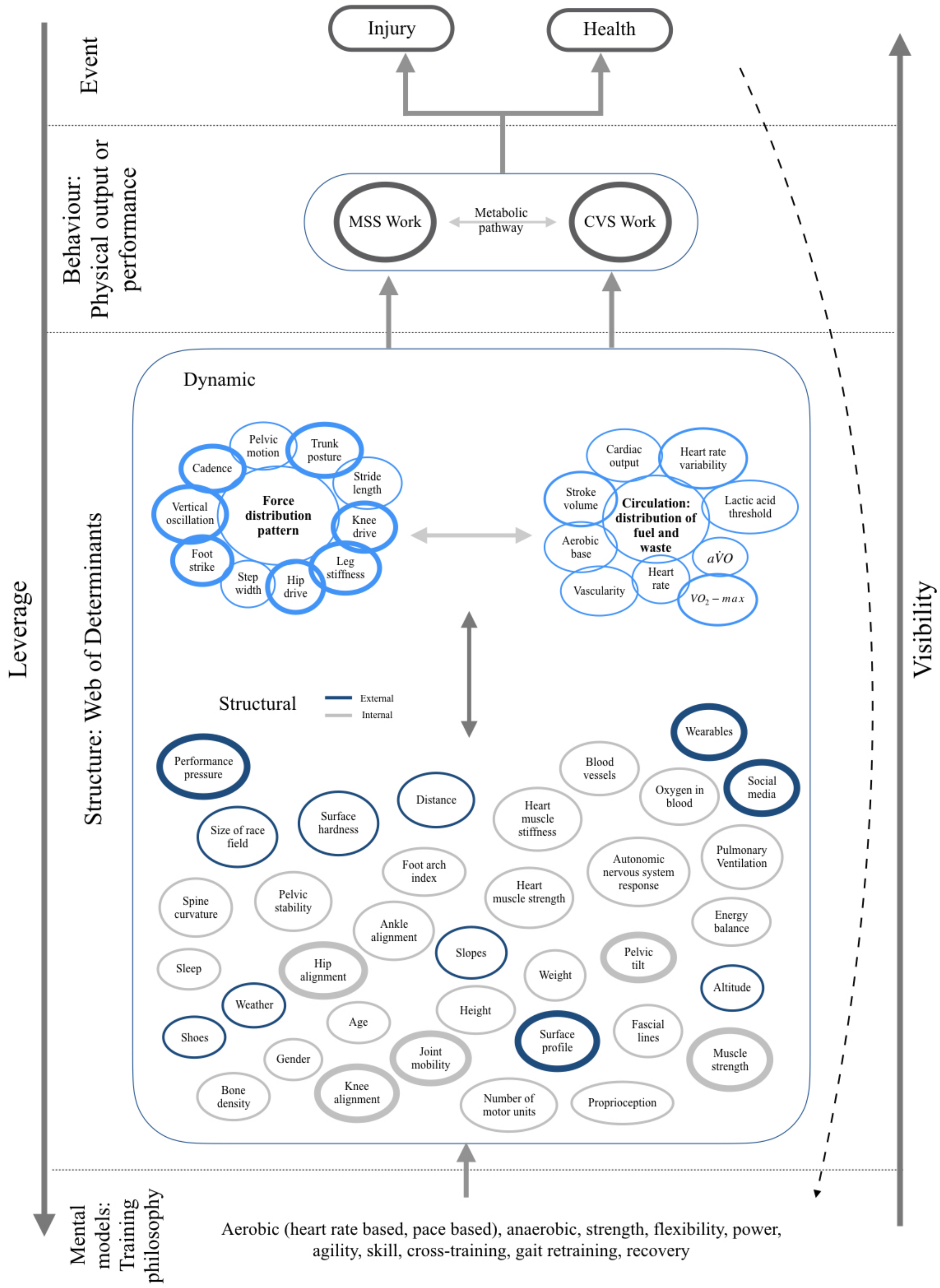


Figure 10.2: The path-to-event as a function of the runner's web of determinants. This figure was published in *Theoretical Issues in Ergonomics Science*, July 2020.

10.1.1 Force distribution patterns

The dynamic web in Figure 10.2 has at its centre the *force distribution pattern*, a profile that results from the interaction between all the dynamic running gait elements. From this presentation, it is clear that studying foot strike pattern in isolation (by ‘keeping all other variables constant’) will not provide the full picture on force distribution patterns. There is therefore a *true* force distribution pattern, and a *what-if* pattern. Some dynamic elements take longer to learn, for instance the forward lean instruction is more difficult for a novice, untrained runner to obtain (even more so during the brief sessions when recordings in the laboratory are made), than simply increasing their stride frequency for a short duration. Even if attempted, the forward lean will not realise efficiently when demanded from an anterior pelvic tilt and stiff hip flexors that do not allow for the range of motion required in the hips and pelvis. With the objective to understand the direct kinetic effects of cadence on lower limb joints in a cohort of runners, a reductionist approach asks “*what if* we increase cadence?” and thereby excludes the interaction with forward lean (by extension to the structural web, also whichever factors are preventing a forward lean) as it might confound the direct effect of cadence *alone*. Another study might only seek the direct effect of a foot strike on the lower limbs (“*what if* the runner lands on the ball of the feet instead of the heel?”), again excluding the other elements that contribute to the *true* force distribution pattern developing throughout the body in the athlete’s real training and running environment.

10.1.2 A locomotion synergy

Locomotion (work done by the MSS) is a function of the extent and intensity scale of the structural and dynamic interactions. The demand for blood placed on the CVS will change between locomotion strategies, with oxygen consumption rising on either side (shorter and longer) of a runner’s desired stride length (Snyder and Farley, 2011). Two basic, collective locomotion strategies to running overground became apparent in the dynamic WoD. First, improve the recycling of the elastic energy from the spring-mass system through a combination of augmented leg stiffness (perhaps at the cost of cadence with a longer stride to increase flight time, subsequently then a larger VO), forward trunk lean, a higher hip drive and mid- to fore foot strike (though a foot strike transition might cause more harm than good (Molloy, 2016)). Second, lowering the negative work in the lower limbs through decreasing VO, higher cadence, and the foot’s ground contact position in relation to the hip at the peak GRF. The elastic recycling approach leans towards a capitalisation strategy (to make the most of what energy is available, colloquial is to say ‘the ground is an ally’) and the second is more defensive, to attenuate load.

10.1.3 Data driven running performance

Wearables (structural-external) that monitor biomechanical variables during running, such as cadence and VO (dynamic), may empower an athlete to alter their locomotion strategy (physical output) towards capitalisation of energy or load attenuation; further aided by freely available on-line coaching on social media platforms (structural-external) that support either strategy. Performance pressure (structural-external) may start to develop when an athlete monitors their metrics and perhaps share online for social support. Driven by the performance pressure to reach their goals, an athlete may lose sight of the structural-internal elements not visible in the data provided by most wearables, such as hip and knee alignments, pelvic tilt, muscle strength and joint mobility. Imbalances in these elements will weaken support for the force distribution patterns as they develop in the dynamic web when cadence and VO are altered. Consequently, the MSS will do work based on a less than optimal force distribution pattern, putting the athlete at risk for a RROI. This information must travel back to the training philosophy to address the structural shortcomings, perhaps through a flexibility and strengthening program.

10.1.4 Environmental influences

Running on a cambered surface profile (external-structural) reinforces knee-toe malalignment (structural), giving rise to an imbalanced hip drive and altered foot strike (both dynamic) by changing the length and orientation of the levers of the lower limbs in relation to the each other. Consequently, the muscles of the lower limb are loaded in a different sequence and perhaps beyond the strengthened range (internal-structural), resulting in excessive muscle strain (Unfried et al., 2013). When the muscle is loaded beyond capacity, a RROI (the event) will follow (Larsen et al., 2016) when work (the physical output) is done by the MSS, but as a product of immoderate force distribution. Furthermore, poor propropception (structural) in the lower limb joints does not allow for timeous feedback to the motor units (structural) to fire and correct the joint alignments through muscular action (structural) (Enoka and Duchateau, 2019); again altering the force pattern in the dynamic web.

Similarly, studying HRV (dynamic) without context of other dynamic determinants in the circulation pattern cannot provide the full picture for CVS integrity in an environment different to the normal training environment. Diminished stroke volume, due to heart muscle stiffness and/or inadequate heart muscle strength (internal-structural), satisfactory circulation of blood during high intensity training at higher altitudes (external-structural) may not initially be possible, despite a favourable resting HRV at lower altitudes and an upper-range VO_2-max (both dynamic) (McArdle et al., 2010).

10.2 The runner's causal loop diagram

The rCLD is presented in Figure 10.3. Main accumulation points in the system are the training pulse, the integrity of structures, and damaged structures. Training pulse is a product of the external-structural elements and the dynamic web of the runner's WoD from Figure 10.2 (§ 10.1 *Describing emergent running performance from interactions using the ice-berg model*): the surface on which running is taking place, training duration, the cadence, leg stiffness and VO. The training pulse may be articulated as: 'to run for t hours with a leg stiffness k at cadence f with a VO of x_m on surface r'. Maturity of structures is a product of the internal elements of the runner's structural WoD (Figure 10.2) aggregated as muscle strength, joint alignment and flexibility. Damaged structure accumulates as a result of higher entropy when the restoration process is neglected whilst the cyclical load on structures continues.

Mainly three balancing and two reinforcing loops characterise the rCLD in Figure 10.3. The first balancing loop, B1, closes the gap between the goal structure and the actual structure of the athlete through the changes in the micro-damage rate (§ 3.3 *Training: a function of biomechanics, physiology and the neuromuscular pathways*). The second balancing loop, B2, also closes the gap between actual and goal structure but through exercise therapy to build new structure. The third balancing loop, B3, removes damage from the athlete's body by some damage control actions taken by the athlete. The first reinforcing loop, R1, amplifies the training pulse of the runner through adjustments in intensity as mature structure closes the gap. The second reinforcing loop, R2, amplifies an unwanted consequence when damage has occurred and is accumulating, but training is not suspended or lowered.

The runner's training cycle in the rCLD reads as follows: *Training pulse* inflicts some micro-trauma to structures (3.3 *Training: a function of biomechanics, physiology and the neuromuscular pathways*), thereby adding to *micro-damage*. *Micro-damage* adds to the internal *entropy* of the RCAS through increased *number of fibres that are now in some state of disarray.* Internal *entropy* adds to the *damage* accumulated by the body, which increases the damage:structure ratio (also influenced by a *limiting factor*). The *damage:structure ratio* (E_n from Equation 5.29 in § 5.2.5 *The entropic line: gauging damage and structural integrity*) represents the relationship between damaged tissue and the state of integrity of tissues. Once this ratio has exceeded a limit from accumulated damage, *cell (structural) death* occurs, representing an injury to structure. The injury takes away from the *integrity of structures* and negates the fitness and health of the RCAS, whereas maturity of structures lowers the damage:structure ratio.

Internal entropy also leads to *restorative actions* taken by the body, of which two modes are differentiated as a function of time: an immediate restorative action by the body after the physical activity to return to homeostasis and a latent component, whereby the body's physiological processes set in to repair micro-damage after the activity. The restoration delay increases with damage build-up and is associated with the environmental temperature and the HR-zone in which training took place. Restored integrity of structures lowers the entropy, and as the runner becomes fitter, the restorative actions stabilise due to the physiological adaptations in the RCAS.

The body's response to micro-damage as a result from the training pulse is to form *new structures* to augment structural integrity to deal with the higher loads imposed by the higher levels of physical activity. *Exercise therapy* through strength and conditioning regimens adds to the new structure formation and reinforces the structural integrity. Mature structure closes the gap between the *structural (fitness) goals* of the runner and the current level of structure, and subsequently the intensity of training may increases, thereby increasing the training pulse. As the gap closes, the training load may increase until the gap is closed and the training load will stabilise.

From the perspective of the underlying growth-and-underinvestment archetype, fundamental solutions to poor structural integrity is captured in the two delays (restorative to maturity and new structures that must mature), adjustments to the training load itself as well as exercise therapy. The symptomatic solutions to negated fitness and minor injuries are captured in the training load, by focussing on adjustments with near instant results, mostly through manipulation of the external-structural web and some gait alternation strategies in the dynamic web.

10.3 The dynamic simulation model

The purpose of the simulation model is not to find a solution, but to identify leverage for long-lasting change. The model's practical focus is on the self-organisation between the spatio-temporal entities' (the natural, external environment) and the runner's internal biomechanical and physiological entities that give rise to the physical output during running and the eventual RROI. The simulation model maps the qualitative rCLD to a quantitative, dynamic version to test the quality of the type of intervention, in terms of longevity of the athlete's structure. These interventions are actions to improve on poor fitness, both long-term and short-term. Long-term interventions focus on fundamental options, characterised by the challenges of the delay between investment and fruition; short-term solutions are actions that have more immediate effect to address poor fitness.

Longevity is studied in terms of the total training load, the level of structure, the damage-to-structure ratio, time spent in damage zones and the time of injury (if it had occurred). Although the calculations in the simulation model are done using Newtonian time $\triangle\ t$, the changes in the structure of the RCAS develop along the unidirectional arrow of time, T (refer to § 5.2 *Entropy and the ambiguity of time in data-driven synthesis of athletic work*).

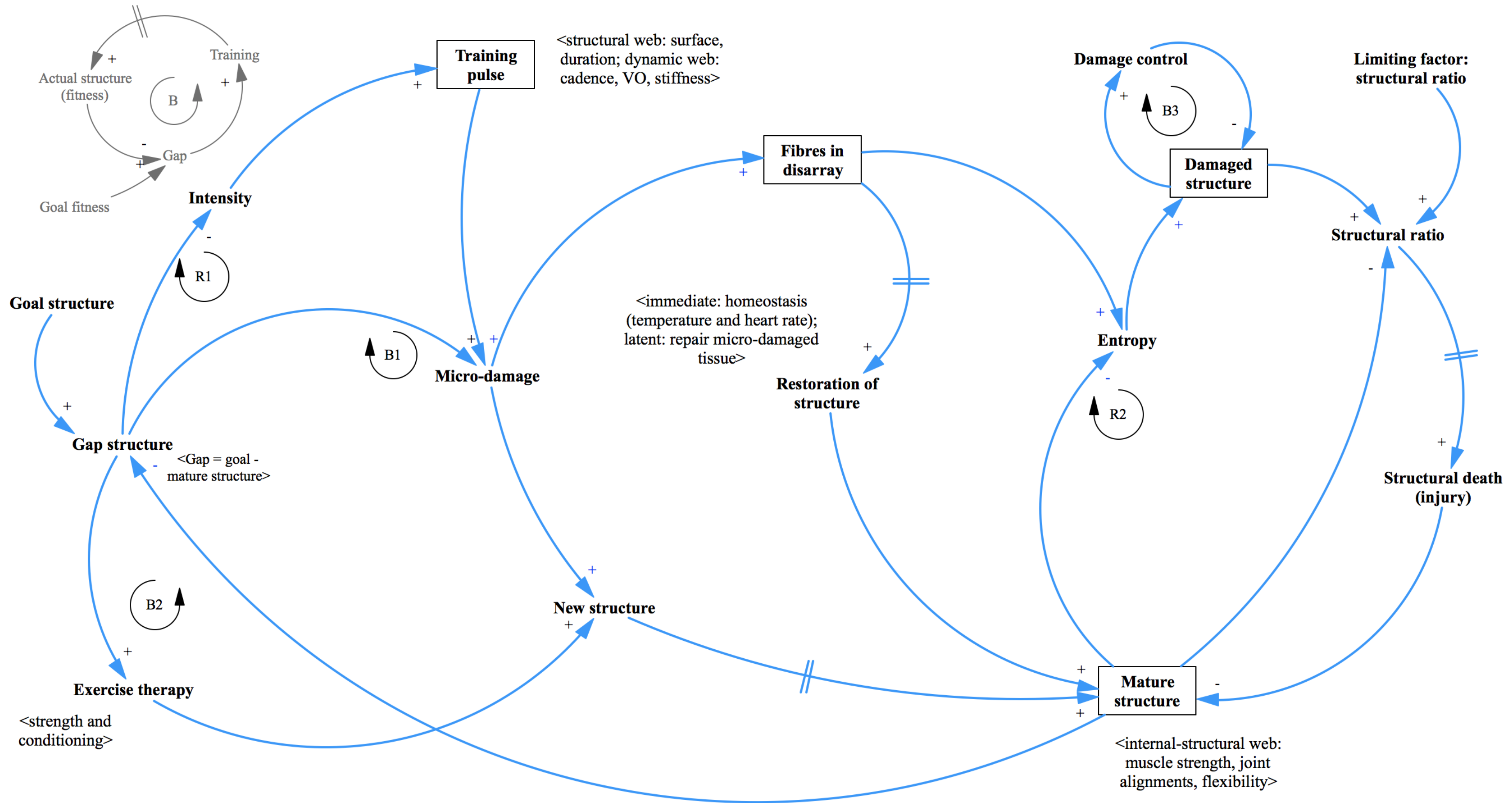


Figure 10.3: The causal loop diagram for the runner

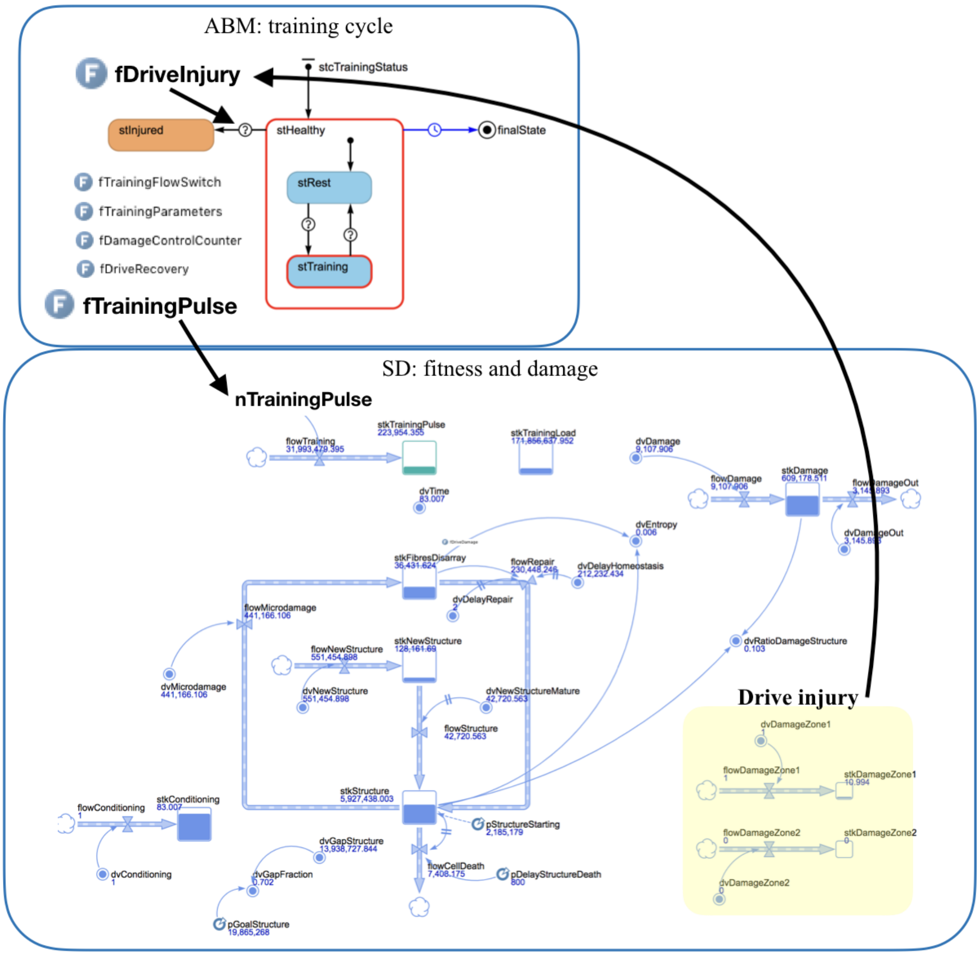


Figure 10.4: Modular components of the integrated hybrid simulation

10.3.1 Hybridisation: agent-based and system dynamic components

The simulation is a hybrid model containing the heterogeneous decision-making elements of ABM in a continuous SD model. The athlete makes individual training decisions through a statechart (the training cycle) and trigger functions. Interactions between the athlete's biomechanical and physiological elements with the environment are modelled on the micro level through functions within the statechart. The accumulation of either fitness or damage in the athlete (the system under study on a macro level) is captured in stocks and facilitated through flows (Figure 10.4). Accumulated damage, in turn, alters the condition of the athlete on the micro level and triggers the statechart into an injury when a threshold is reached. The athlete as an agent is allowed to alter their training load during the run of the model as the structure moves through demarcated fitness and damage zones.

The behaviour over time and cluster analyses (§ 8.2 and § 8.3) pointed to micro level interactions between the surfaces encountered, cadence, and HR. Interactions between the surface, leg stiffness, and VO are based on literature (§ 4.7 *Environmental stressors in running*), specifically the runner as a spring-mass model (§ 5.1.2 *Modelling the spring-mass system with simple harmonic motion*). Interaction between temperature and recovery were found in the literature (§ 3.1.4 *Thermoregulation*). The agent-based component abstracts the interactions between leg stiffness, VO, cadence, HR, temperature, and surface type (through the run type). Interactions between the athlete, the altitude, and the direction of the slope are not included in the simulation, but are suggested as focus areas for future work.

The stock-and-flow model in Figure 10.6 is the quantified, dynamic abstraction from the qualitative rCLD in § 10.2 *The runner's causal loop diagram*, displayed in Figure 10.3. The main aspects of the model is discussed here, for more detail the reader is referred to the simulation documentation containing all of the model's elements and their properties, in Appendix D. The stock-and-flow model essentially represents the RCAS as a closed plumbing system (recall from § 6.4.2 *The stock-and-flow simulation model*), within which pressure points develop as a result of unwanted accumulation of damage. Actions taken by the athlete (governed through the decision rules in the ABM component) opens and closes the valves (flows) into and out of the accumulation tanks to relieve pressure or divert content.

In the *Anylogic* simulation model, various objects with built-in functionalities are used to perform a certain task in the running model. The endogenous elements from the WoD and the rCLD have been assigned to types of variables in the simulation model, with the assigned *Anylogic* symbol as follows:

- Accumulating variables are captured in stock or levels (for example, the structure, damaged structure, training load and days spent in damaged zones).
- The parameters are measurable factors to define some numerical system attributes. They are set at start-up and update during the simulation when the functions in which they are set, are called into action. Parameters are unique attributes from the WoD and drive the accumulation or dissipation of structure, damage and training loads through rates (flows).
- The flow rates may consist of mechanistic models, such as exponential functions and/or waveform functions.
- Variables are mostly used to facilitate dynamic parameter setting as the model runs and the athlete makes decisions regarding training.
- Dynamic variables are a special object that update automatically with time and are driven by return functions. Dynamic variables were needed to code the dynamic adjustments for certain flows as the model runs through differentiated damage and fitness zones.
- Collections are used to store input data from which parameter settings are sampled.
- A function is the space where custom code is written to drive dynamic variables, and/or alter parameter or variable values according to rules.

10.3.2 The training-rest cycle

The statechart `stcTrainingStatus` models the athlete's transition between resting and training (Figure 10.5). The statechart is nested: at the highest level, the athlete is either healthy (in state `stHealthy`) or injured (in state `stInjured`). The lower sub-levels of `stHealthy` consist of active training (`stTraining`) or at rest (`stResting`). Only while in the healthy status may an athlete train, once injured the training stops. The transition to `stInjured` is modelled as a condition, which is based on the time spent in the damage zones (see § 10.3.6) and managed by a return function, `fDriveInjury`. The entire simulation allows for 720 days of transitioning between training and resting, before transitioning to the final state and the simulation ends.

The athlete transitions to `stTraining` when the function `fTrainingFlowSwitch` is activated. Once the function `fTrainingFlowSwitch` is deactivated, the athlete transitions back to `stResting`. The `fTrainingFlowSwitch` is activated based on time elapsed since the previous session ended and the interval time between training sessions. The next session will start 23 hours after the last session ended and then last for 0.0417 days (one hour) in the base model.

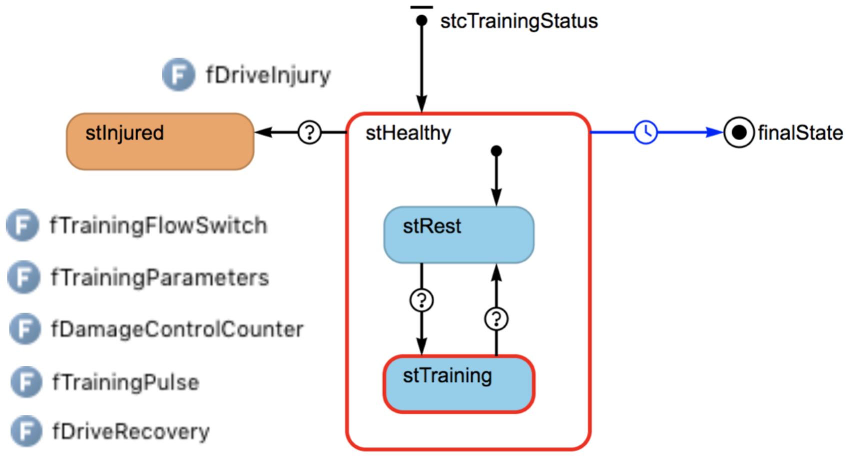


Figure 10.5: The training status statechart

This interval time may be adjusted during experimentation, but the interval is based on the athlete's frequency of interactivity times from Table 8.3, where it was shown that the majority of activities start within 24 hours of the last activity. The statechart records the time a session has ended, re-setting the clock in `fTrainingFlowSwitch`. The functions `fTraining-Parameters`, `fTrainingPulse`, `fDamageControlCounter` are all called upon in the `stRest` to enforce the runner's decision rules for the upcoming training session and to deal with damage (§ 10.3.6). Training rules for parameters are coded into the function, `fTrainingParameters`. The surface on which the run takes place, the associated cadence, VO, leg stiffness, HR, and the temperature are sampled in this function. The function `fTrainingPulse` calculates the training load for that session (see § 10.3.4 for the mathematical detail).

Recovery rules are coded into a return function, `fDriveRecovery` and adapts staggered parameters for each damage zone (§ 10.3.6). Function `fDriveRecovery` is called upon in a continuous dynamic variable, `dvDelayRepair` to manage recovery and conditioning over time, whereas `fTrainingParameters` are called upon before the athlete transitions to training.

10.3.3 The stock-and-flow diagram of the runner

Figure 10.6 contains the detailed stock-and-flow diagram of the simulation. (Not all the arrowed lines in the figure are functional as per traditional stock-and-flow configurations. In the *Anylogic* computational configuration, arrows between driving functions, , and parameters, flows, stocks, and variables are not necessary. However, they are shown in the figure to provide the reader with a visual of the closed-loop, 'pressurised plumbing system'.)

Once per day, the training flow, `flowTraining` is activated to pulse the wave function that characterises the spring-mass system of a moving runner. The function, `fTrainingPulse` calculates the size of the pulse before the training session starts (see § 10.3.4). The `flowTraining` is activated and deactivated by the function `fTrainingFlowSwitch`, which monitors the position of time in the simulation as to when each training session starts and ends. The total, positive impulse generated during the training session is absorbed as the training load, into stock `stkTrainingLoad`. During the training session, the flow `flowMicrodamage` is activated; this flow removes fibres from stock `stkStructure` to the stock for temporarily disarrayed fibres, `stkFibresDisarray`. Its magnitude is proportional to the training impulse in `flowTraining` by the nominal micro-damage rate, `pNominalMicrodamageRate`. Micro-damage is coupled to the fitness gap, and as the gap closes, the flow `flowMicroDamage` decreases:

$$\texttt{flowMicroDamage} = \frac{\texttt{dvGapStructure}}{\texttt{pGoalStructure}} \times \texttt{pNominalMicrodamageRate} \times \texttt{flowTraining} \tag{10.1}$$

At a gap of 0.005, Equation 10.1 reduces to only an adjusted nominal rate, to prevent a negative rate when the gap is breached.

There is a delayed return flow, `flowRepair` from stock `stkFibresDisarray` to stock `stk-Structure`. The `flowRepair` is additive, consisting of short term homeostatic reactions immediately following exercise, and longer term repairing of micro-damage delays. The dynamic variable for the delay in homeostasis, `dvDelayHomeostasis`, is calculated as the function of the training duration, the external environmental temperature, and the HR-zone of the exercise bout:

$$\texttt{dvDelayHomeostasis} = \texttt{pTrainingDuration} \times (0.005 \times \texttt{pTemperature} + 0.02 \times \texttt{pHeartRateZone}) \tag{10.2}$$

Higher environmental temperatures and training in higher HR-zones therefore prolong the recovery delay. An one-hour exercise bout in an environment with a temperature value of 20° and a HR-zone of five would equal to a homeostasis recovery delay of 4.8 hours, which is consistent with the reported natural recovery time of four- to five-fold the exercise time in Kenny and McGinn (2017). The athlete's mean HR of 144 *bpm* forms part of zone 3 and the average temperature for the year during the exercise times is 18.95°C. These parameters would result in a 3.7-hour recovery time, still close to the recovery time of between 4 and 5-fold the exercise time.

The longer-term reaction involves repairing micro-damaged fibres over time, representing the recovery phase of training. This delay time is presented as a dynamic variable, `dvRepair` and is re-set in the function `fDriveRecovery` according to the damage zone. The `flowRepair` can now be written as:

$$\texttt{flowRepair} = \frac{\texttt{stkFibresDisarray}}{\texttt{dvRepair} + \texttt{dvDelayHomeostasis}} \tag{10.3}$$

New structure (example, muscle growth) is formed by the body in `stkNewStructure` stock. Inflow to `stkNewStructure` is driven by a dynamic variable, `dvNewStructure`, which in turn, is a function of `flowConditioning` and `flowMicrodamage`. New structure takes time to mature, before it moves through the delayed flow `flowStructure`, into `stkStructure`.

The path to injury is a two-step process. First, net entropy (in `dvEntropy`) is a fractional relational measure between temporary disarrayed fibres (internal entropy), as a result of micro-damage from training, and existing structure. This calculation is the first pressure point, derived from the entropic line gauge in § 5.2.5 *The entropic line: gauging damage and structural integrity*):

$$\texttt{dvEntropy} = \frac{\texttt{stkFibresDisarray}}{\texttt{stkStructure}} \tag{10.4}$$

The `dvEntropy` updates continuously to keep track of the relationship between the number of fibres in disarray and structural integrity. At some threshold, e_t, a portion of fibres in disarray is considered damaged and is then added to the damage stock at a rate of α_d%.

As such, `dvEntropy` controls the flow into the stock for damaged structure, `stkDamage`. In the second step towards injury, when `dvEntropy` reaches a certain threshold, the `flowDam-age` is activated and takes as value a fraction of the `stkFibresDisarray`. The outflow from stkDamage, flowDamageOut, is additive, accounting for natural removal of damaged tissue and damage control measures taken by the athlete.

The second step to injury involves the cumulative measure of damage: the $^d/_s$-ratio considers the amount of damaged fibres to the amount of structural integrity, and serves as a measure to gauge the cumulative damage from overload. This is the second pressure point in the plumbing system. Two damage zones exist, with the upper and lower limits for each zone demarcated using the $^d/_s$-ratio. An athlete may spend some time in these damage zones; however, it is the cumulative time in the damage zone that will ultimately incite the RROI. The damage zones are defined as lower and higher risk. The fractional ratio between the two stocks, stkStructure and stkDamage make up the dynamic variable dvRatioDamageStructure; this is the $^d/_s$-ratio:

$$^d/_s\text{-ratio} = \texttt{dvRatioDamageStructure} = \frac{\texttt{stkDamage}}{\texttt{stkStructure}} \tag{10.5}$$

The dvRatioDamageStructure controls the flow of the damage zone drivers through associated dynamic variables: either flow, flowDamageZone1 or flowDamageZone2, depending on the magnitude of dvRatioDamageStructure, is activated and flows at a rate one unit-day per simulation-day to add to the stock for the damage zones (stkDamageZone1 or stkDamageZone2). An injury is incited when a certain number of days has accumulated in the damage zone stocks, see § 10.3.6. An injury is incited by instantaneous emptying the content of stkStructure: accelerating the flow of cell death, flowCellDeath by shortening the delay, pDelayStructureDeath to one day.

The gap between stkStructure and the goal structure, pGoalStructure, is continuously updated in the dynamic variable, dvGapStructure, and is calculated as the difference between them: dvGapStructure = pGoalStructure − stkStructure. The proportion of the closed fitness gap, dvGapClosedFraction, drives the progression through the fitness zones, see § 10.3.7.

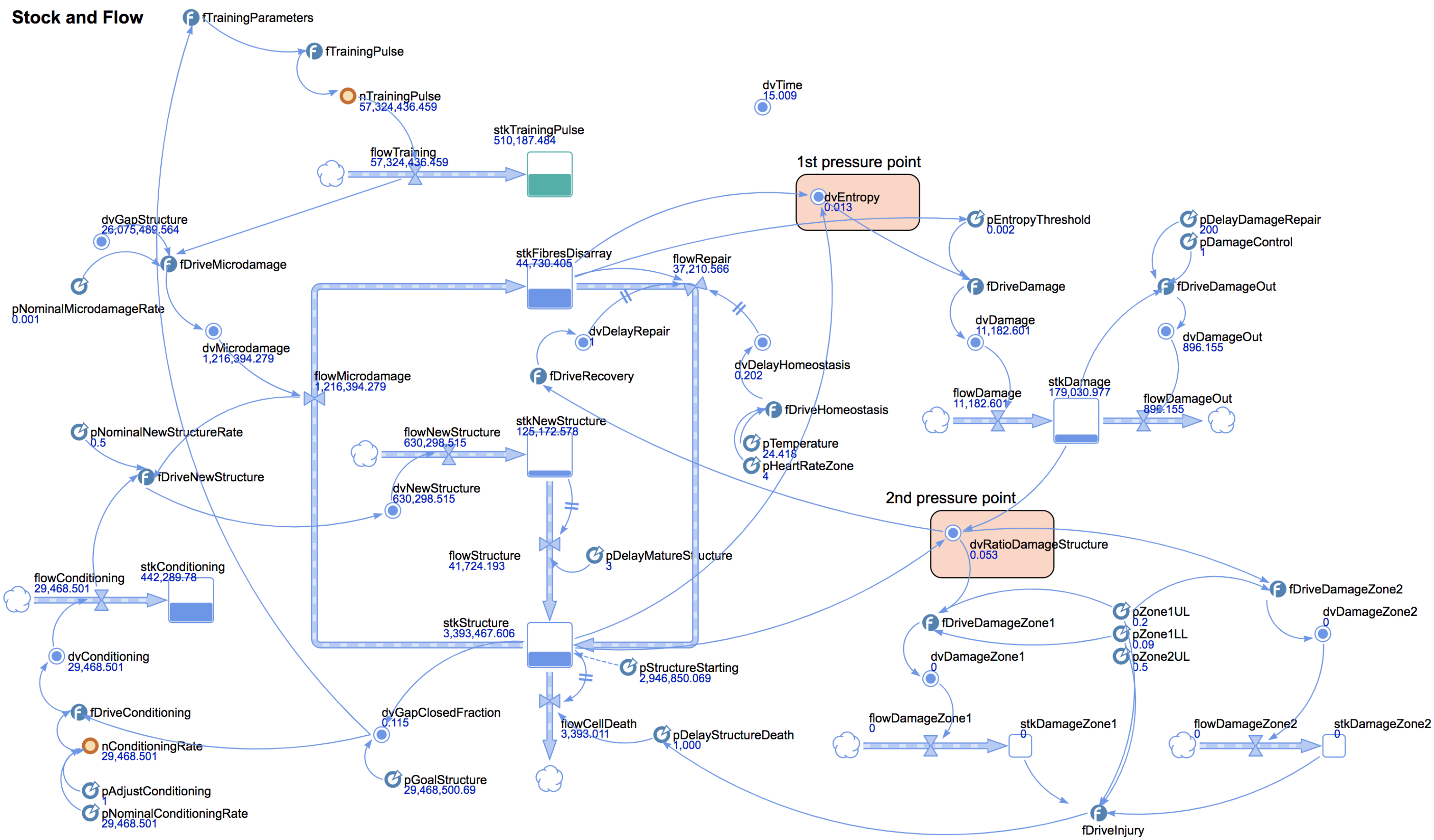


Figure 10.6: The detailed stock-and-flow model of the runner

10.3.4 Selecting training parameters and the main input function: pulsed training load

Training input parameters are set before the start of the training session and generally follows the sequence in Algorithm 1. The training parameters that are assigned according to the surface type are the cadence, leg stiffness, and VO (from Table 10.4). Heart rate is sampled from the classification framework (Figure 10.7), following the steps in Algorithm 2.

Algorithm 1: Setting up parameters for the training session

```
for each new training session do
    Randomly select the run type j from the PMF (Table 10.5)
    Assign training parameters according to run type distributions (Table 10.2)
    Adjust training intensities, depending on fitness zone (§ 10.3.7)
    Set training time
    Randomly sample temperature values based on month of the year
    Calculate total training pulse (Equations 10.7 and 10.8)
end
```

Algorithm 2: Setting up heart rate from cadence

```
for each cadence selection do
    assign cadence (f_j) to a level (low, mid, high) using Equation 10.6
    randomly select the HR level by evaluating the PMF for HR levels, Table 10.1
    assign the randomised HR parameters as per Figure 10.7
end
```

$$\text{Cadence level} = \begin{cases} \text{low} & : f_j < 3.0 \\ \text{mid} & : 3.0 \leq f_j < 3.167 \\ \text{high} & : 3.167 \leq f_j \end{cases} \tag{10.6}$$

Table 10.1: Probability mass function for heart rate and cadence levels

Cadence level	**Heart rate level**	**Probability**
Low	Low	0.668
	High	0.332
Mid	Low	0.3897
	High	0.6103
High	Low	0.3216
	High	0.6784

The runner is represented as a single spring-mass model undergoing SHM (refer to § 5.1.2 *Modelling the spring-mass system with simple harmonic motion*). In the base model, the stimulating input for training, the `flowTraining`, is pulsed for one hour each day. That is, the `flowTraining` in the simulation is only active for one hour per day. The flow quantity for `flowTraining` is calculated as a single, aggregated impulse value in the variable `nTrainingPulse`.

To calculate a single impulse value for `nTrainingPulse`, the actual training time is subdivided into smaller time steps to account for instantaneous forces. The instantaneous force as

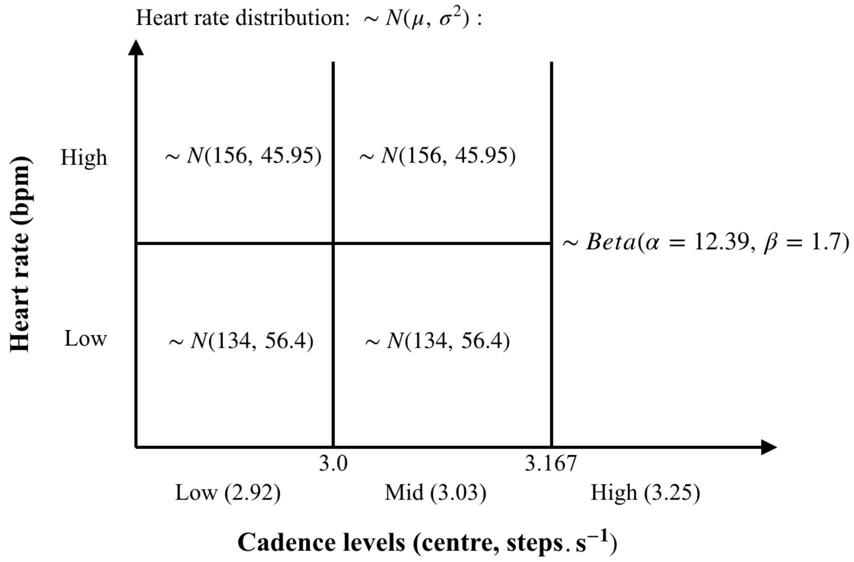


Figure 10.7: Parameters for heart rate from cadence classification

encountered at time step i is calculated in Equation 10.7 (derived from the SHM waveform of the runner as a spring-mass model in Equation 5.27):

$$f(t_i) = -k_v \times x_m \times \cos(2\pi\ f_j\ t_i) \tag{10.7}$$

where: k_v is vertical leg stiffness, x_m represents the amplitude and f_j is the frequency (cadence) in steps.s^{-1} for surface encountered on run type j. The vertical leg stiffness k_v is assigned various values from a realistic range obtained from Ferris and Farley (1997). x_m is the amplitude, or the VO of the CoM of the runner, also assigned ranges from Ferris and Farley (1997). Furthermore, $t_{i+1} = t_i + \triangle\ t$, where the time step $\triangle\ t = 0.001$ and $i \in [0, T_n]$. T_n is the total number of time intervals to complete and is calculated using the total number of seconds in an hour, 3600, divided by the time step $\triangle\ t$: $T_n = {}^{3600}/_{\triangle\ t}$. Figure 10.8 shows the force $(f(t_i))$ waveform of the runner as a spring-mass model for one second of running at a cadence of 2.991 steps.s^{-1}, a VO of 0.098 m and vertical leg stiffness of 17.6 kN/m.

The total impulse is calculated in Equation 10.8 (following the training load function as presented in Equation 5.28), as the sum of the absolute value of $f(t_i)$ over $i \in [0, T_n]$ multiplied by an $\alpha = 0.5$, since only half of the total impulse is associated with ground contact (stance phase) during which energy must be absorbed into the body and preferably elastically recycled.

$$J = 0.5 \times \sum_{i=0}^{i=T_n} |f(t_i)| \tag{10.8}$$

J is therefore the total training load pulsed within one hour and assigned to the variable `nTrainingPulse`. In order to allow the running model to only pulse J within the hour, the `flowTraining` is set at ${}^{J}/_{0.0417}$, where $0.0417 = {}^{1}/_{24}$. When the flow for training is activated, the `flowTraining` will be equal to ${}^{J}/_{0.0417}$, yet at the end of the training hour, the total load pulsed will be equal to J.

Mentioned earlier in § 8.3 *Cadence*, the mechanism for track training is slightly different. In the simulation model, when sampling from the higher cadence group (3.24 steps.s^{-1}), an attempt was made to pulse the function for the proportion of time the athlete is actively training at faster paces and higher cadences, which amounted to 53% of the total training time at this higher cadence. In the simulation, the `flowTraining` is multiplied by 0.53 to only

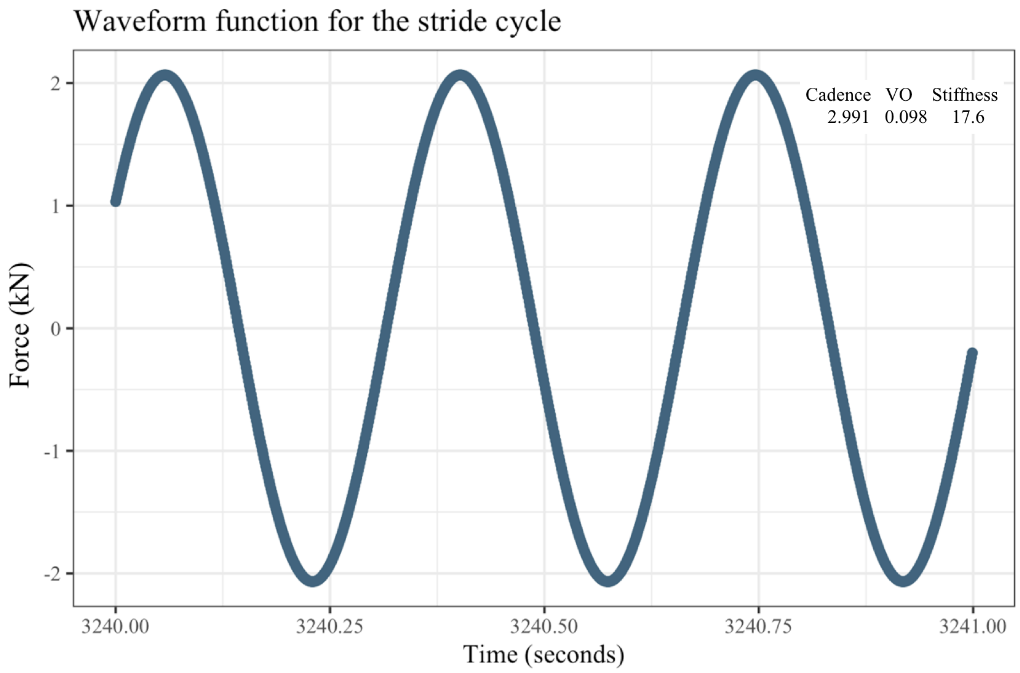


Figure 10.8: The waveform function for the runner as a spring-mass model

allow a training activation of 53% of the hour. By considering different values for the runner's biomechanics across varying terrain, the training load is therefore a mathematical configuration of the spatio-temporal interaction between the elements of the WoD of the RCAS.

10.3.5 Structure and damage

The most important points of accumulation in the system are the stocks for structural integrity, `stkStructure` and damaged structure, `stkDamage`. Their relationship is modelled as the $^{d}/_{s}$-ratio. The $^{d}/_{s}$-ratio represents the extent of the damage in relation to structural integrity. This ratio indicates the pressure that the RCAS is experiencing and becomes a pivotal point in the simulation, whereby decisions are made regarding training load, scaling action to release damage, and the inciting of an injury, based on some critical values. Bittencourt et al. (2016) referred to a system's critical values as those values of the system's elements that when reached, the elements configure in such a way to produce patterns or regularities. The function, `fDriveRecovery`, re-sets all delay and response parameters, using the magnitude of the $^{d}/_{s}$-ratio and its position within the damage zones (Equation 10.9).

The `stkStructure` is a collective variable to include all the endogenous biomechanical and physiological variables in the runner's WoD (refer to Figure 10.2, § 10.1). Integrity of biomechanical and physiological variables is protective against injury. The initial structure (starting value) assumes 120% capacity to absorb the total training pulse, J, of the first training session.

10.3.6 Progressing through the damage zones

Damage zones represent an aggregated status of the $^d/_s$-ratio. Four damage zones are demarcated along its critical values:

$$\text{Damage zone} = \begin{cases} 0 & : {}^d/_s \leq 0.09 \\ 1 & : 0.09 < {}^d/_s \leq 0.2 \\ 2 & : 0.2 < {}^d/_s \leq 0.5 \\ 3 & : {}^d/_s > 0.5 \end{cases} \tag{10.9}$$

Zone 0 represent minimal damage, healthy and manageable conditions, zones 1 and 2 are lower- and high risk zones respectively, and zone 3 is permanent damage. These zones were calibrated using sensitivity analysis and extreme condition tests. The lower limit for zone 1 was found using an extreme starting condition for `stkStructure`. The athlete should have entered the damage zone almost immediately at a starting `stkStructure` value of 10% of the initial (first) `flowTraining`, which occurred at a $^d/_s$-ratio of 0.09.

The function, `fDriveRecovery` facilitates the actions taking place as the athlete progress through the damage zones. Once the athlete enters damage zones 1 and 2, the following occur:

1. the `flowRepair` slows down;
2. the natural rate of cell death, `flowCellDeath`, increases;
3. the athlete makes decisions regarding training load adjustments.

In both instances (1) and (2), the compounding effect of overuse burden is modelled: the `stkStructure` is not replenished fast enough to anticipate the next load, whilst simultaneously losing more fibres as part of natural cell death. The denominator of the $^d/_s$-ratio is therefore becoming smaller, resulting in a higher $^d/_s$-ratio, keeping the athlete in the damage zone they are in or driving them into the higher risk damage zone.

When the athlete enters a damage zone, they may decide to alter their behaviour through training load adjustments and utilise other short-term damage control mechanisms. The damage control mechanisms will drive the `flowDamageOut` to lower the stock `stkDamage`, in an attempt to lower the $^d/_s$-ratio and reverse out of the damage zones. However, the damage control mechanism is subject to mechanical wear, and was modelled as a hyperbolic function with declining efficacy over time. The variable `nCounter` started at a value of 1 and increased by a margin of 0.01 for each training session (in function `fDamageControlCounter`). The parameter for damage control (in damage zones 1 and 2) is then divided by the `nCounter` to model the degrading efficacy of the mechanism:

$$\texttt{pDamageControl} = \frac{\texttt{pDamageControlZone1}}{\texttt{nCounter}} \tag{10.10}$$

In this model, after 100 continuous days of use, the damage control mechanism is only half as effective as it was initially (since `nCounter` = 2 after 100 days).

An injury is incited based on the number of days accumulated in the damage zone stocks. The athlete is allowed more time in the lower risk zone before an injury occurs (42 days), whereas the higher risk zone allows for less time before the injury occurs (10 days). A buffer was created to prevent migration to the higher risk damage zone when the remaining time in the lower zone was short enough to lead to an injury before the maximum time in the higher zone would have been reached. When the remaining time in the lower damage zone was less than or equal to 10 days, the injury would be incited instead of migrating to the higher risk zone.

10.3.7 Progressing through the fitness zones

Fitness zones are demarcated by the size of the closed fitness gap in relation to the goal. The closing of the fitness gap, z_f, through a dynamic variable, `dvGapClosedFraction` is simply the level of structure, `stkStructure` divided by the goal structure, `pGoalStructure`:

$$z_f = \texttt{dvGapClosedFraction} = \frac{\texttt{stkStructure}}{\texttt{pGoalStructure}} \tag{10.11}$$

As the gap closes, the proportion z_f approaches a value of one. Four zones are demarcated at the critical values (cut-off points) $\{0.25, 0.5, 0.75\}$, where:

$$\text{Fitness zone} = \begin{cases} 0 & : z_f < 0.25 \\ 1 & : 0.25 \leq z_f < 0.5 \\ 2 & : 0.5 \leq z_f < 0.75 \\ 3 & : z_f \geq 0.75 \end{cases} \tag{10.12}$$

As an athlete progress through the zones, they may incrementally increase their training pulse through longer training durations. Zone 0 marks early progression of fitness and the athlete trains at the starting parameters. In one experiment, the training pulse was increased by a 10% increase in the training duration for every fitness zone.

10.3.8 Conditioning

An athlete may decide to engage in other strengthening exercises to improve on their structural integrity. A nominal conditioning rate adds new structure at 1.2% of the initial training pulse, `flowTraining`, per day. Conditioning also depends on the fitness gap, with increases in intensity as the gap closes with adjustments made according to the fitness zones (Equation 10.12) in the return function, `fDriveConditioning`.

10.3.9 The interventions to augment fitness

The athlete makes decisions regarding training load and damage control in order to augment their fitness. Interventions are characterised as fundamental or symptomatic. Fundamental solutions have a longer-term focus and are subject to delays, whereas symptomatic solutions immediately address the problem (they act as quick fixes) but loses efficacy over time. Examples are as follows:

- Fundamental: increased conditioning, slower ramp-up of training load, reduce training load once in damage zone 1 or 2, and respecting the repair delays by increasing the intervals between training sessions.

- Symptomatic (i.e. quick fix): increasing damage control, increasing the training load.

Damage control is a collective term for any action that an athlete takes to address shortcomings or training induced damage, in the real world this would include orthrotic braces, compression wear, new shoes and so forth. These interventions vary in successful outcomes, but were grouped together since the basic behaviour of the solution remains the same, irrespective of the mechanism: damage will be removed starting immediately. In the model, damage control is therefore dimensionless, and articulated as activities that remove x units of damaged fibres per day. It is represented by a parameter, `pDamageControl`, which updates in `fDriveRecovery` and is subject to the damage zone (Equation 10.9).

Table 10.2: Input training parameters

Parameter (`name in Anylogic`)	**Expression in *Anylogic* and units**
Primary data	
Surface (`pSurface`)	$j \in \{rc, rr, tc, tr, tt\}$
Cadence (frequency) (`pFrequency`)	$\sim N(\mu_j, \sigma_j)$ steps.s^{-1} for surface j
Heart rate (`pHeartRate`)	Follow classification framework, Figure 10.7
Interactivity time (`nTrainingIntervalTime`)	0.958 days (23 hours)
Temperature (`pTemperature`)	$\sim N(\mu_m, \sigma_m)$°C for month m
Secondary data	
Vertical oscillation (amplitude, x_m) (`pXm`)	$\sim N(\mu_j, \sigma_j)$ meter for surface j
Leg stiffness (`pStiffness`)	$\in \{20, 20, 30, 30, 40\} kN/m$ for surface j
Illustrative data	
Training duration (`pTrainingDuration`)	1 hour per day: 0.0417 days
Nominal conditioning (`pNominalConditioningRate`)	29469 (unit-less)
Fitness zone limits (upper and lower)	zone 1: $(0.25, 0.5)$ zone 2: $(0.5, 0.75)$

10.3.10 Simulation input parameters and variables

Tables 10.2 and 10.3 contain the parameters for the simulation. The actual running type, cadence and HR are mined from the participating athlete's RW data. Table 10.5 contains the PMF for the various running types from which the simulation input is sampled. The running type is used as a proxy for the surface. The collection of running types (rc - road racing, rr - road running, - tc - trail racing, tr - trail running, tt - track training on grass) are the starting point from where the other running parameters are set up based on literature and empirical work. Table 10.4 contains the input values mined from the RW for the simulation model parameters. The cluster analyses from § 8.3 served as a guide to ensure the sampling was representative of the real world behaviour and not a blunt approach. With this guidance from the clusters, the track training subset is split into two values for cadence, namely high and low to represent the bi-modality in the data according to a PMF that distinguishes between the clusters.

The values for VO and leg stiffness interact with the type of surface encountered (§ 4.7 *Environmental stressors in running*) during the run. Data ranges from Ferris and Farley (1997) were used as sensible estimates for these parameters. The nominal conditioning rate is set at 1.2% of the initial training impulse.

Structure and damage management are executed in `fDriveRecovery`, using parameters set up in Table 10.3. These values were calibrated during sensitivity analysis and extreme condition tests.

10.3.11 Data output from the simulation

The discrete output at end of the simulation is recorded to an Excel file, as a set of variables for analysis and trade-off. These variables are the actual gap size, fractional gap (gap as a fraction of the goal), time when the gap was closed (if applicable), time of injury (if applicable), total training load, total time in damage zones (if applicable).

Datasets that are more granular are also captured: once per training session and per half-day. The per-training-session dataset included training pulse, surface type, majority of the HR that training was performed in. The $^d/_s$-ratio and entropy are recorded at half-day intervals for a more continuous evaluation over time.

Table 10.3: Illustrative input parameters for structure management and delays

Parameter (`name in Anylogic`)	**Expression in *Anylogic* and units**
Delay repair per damage zone (`pDelayRepair`)	$\in \{1, 1.5, 2\}$ days
Delay mature structure (`pDelayMatureStructure`)	3 days
Nominal new structure rate (`pNominalNewStructureRate`)	0.5 (unit-less)
Nominal micro-damage rate (`pNominalMicrodamageRate`)	0.001 (unit-less)
Delay: natural cell death per damage zone (`pDelayStructureDeath`)	$\in \{1000, 800, 800\}$ days
Entropy threshold (`pEntropyThreshold`)	0.002 (unit-less)
Damage zone limits (upper and lower)	zone 1: $(0.09, 0.2)$ zone 2: $(0.2, 0.5)$
Damage control per damage zone (`pDamageControl`)	$\in \{1, 4000, 5000\}$ (unit-less)
Increase training duration (`pIncrTrainingDurationSwitch`)	$\in \{0, 1\}$ (unit-less)

Table 10.4: Input values for parameters per surface type

Run (surface) type j	**Cadence: mean ± st.dev** (steps.s^{-1})	**VO: mean ± st.dev** ($meter$)	**Leg stiffness** k_v (kN/m)
Road race (rc)	3.038 ± 0.080	0.12 ± 0.012	20
Road running (rr)	2.981 ± 0.077	0.12 ± 0.012	20
Trail race (tc)	3.075 ± 0.059	0.08 ± 0.008	30
Trail run (tr)	2.953 ± 0.095	0.08 ± 0.008	30
Track training (tt)	low: 2.926 ± 0.0605	0.06 ± 0.006	40
	high: 3.244 ± 0.0712	0.06 ± 0.006	40

Table 10.5: Input probability mass function for run types

Run (surface) type	**Probability**
Road race (rc)	0.003
Road running (rr)	0.399
Trail race (tc)	0.006
Trail run (tr)	0.154
Track training (tt)	0.438

10.4 Conclusion

The qualitative modelling of the runner and the quantified hybrid simulation model were unpacked in this chapter. The hybrid model is able to handle micro level individual decision-making by the athlete at discrete time points in the simulation and based on system level characteristics, although this posed challenges to maintain the continuous time scale of the aggregated SD component. The main input function to pulse the training load in the agent-based component to start the SD component and the high-level progression of accumulation in the stock-and-flow model are both based on principles from physics, although on different levels of abstraction. The primary input data to the simulation model, mined from the RW, makes the model to be subject-specific, although it is possible to extract data from a different athlete and adjust the model accordingly.

Part III

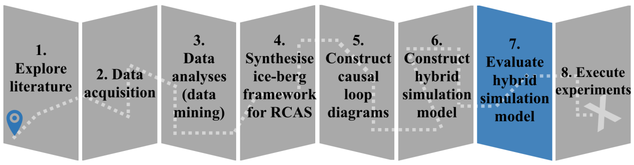
Demonstration and evaluation

Chapter 11

Evaluation of the simulation of the runner as a complex adaptive system

Contents

This chapter formulates task 7 from the research methodology.



The simulation of the RCAS is evaluated for its contribution to research following the DSR assessment by Hevner et al. (2004) and the model tests from Sterman (2000):

1. *Existing artefacts and the new artefact*: purpose and shortcomings.
2. *Adequate mapping of the artefact to the real world:* boundaries and structure assessment, parameters, and dimensional consistency.
3. *Capability of artefact to solve the problem:* usefulness, shortcomings, time horizon, level of aggregation, and system improvement.
4. *Demonstration of the artefact:* running costs, integration error, sensitivity analysis, and extreme conditions tests.

Although SD cannot be fully validated, it remains important to develop confidence in the model as the most applicable version of the abstracted reality to base decisions upon. Models can and should be tested beyond their design limits to ascertain their useful domain (Wakeland and Hoarfrost, 2005). The model testing principles as per Sterman (2000) and Forrester and Senge (1980), are used to evaluate the simulation model, within the framework of DSR.

11.1 Existing artefacts and the new artefact

No other artefact could be found where the basic relationship between structural fitness and overuse injuries was modelled as a pressurised plumbing system, using ST and a hybrid SD and agent-based model. A SD simulation is not an analytical solution, but provides projections, which are imprecise and conditional, of model behaviour over time (Starr, 1980).

The simulation model forms part of a framework to instantiate practical ST in sport science. The purpose of the model is to facilitate practical hypothesis testing from the qualitative rCLD, which is based on three system archetypes. The model was based on system archetypes, with the agent as an athlete who may decide what path to solution they will follow to remain injury free whilst improving their fitness. The model demonstrated the behaviour of how quick fixes and shifting-the-burden strategies culminate in growth-and-underinvestment. To support reproducibility of results, the input parameters and zone demarcation limits were documented for various simulation runs with parameter variations in order to compare output of each unique set of input variables.

11.2 Adequate mapping of the artefact to the real world

The boundaries, structure assessment, parameters, and dimensional consistency (Sterman, 2000) are considered to evaluate the mapping of the simulation model to the real world.

11.2.1 Boundaries and structure assessment

To model the structure dynamically, the endogenous elements from the WoD are differentiated into variable types in the simulation model: levels (stocks), parameters, facilitating variables, dynamic variables, and rates (flows). The simulation model is driven by real-world data generated from athletes' RW as well as environmental data from their training space (weather and geography). Endogenous to the system are the elements in the WoD from Figure 10.2. The WoD was published in the international journal *Theoretical Issues in Ergonomics Science*, which provided confidence in the structure of the framework. The boundary of the system was drawn outside of the runner, to include some of their immediate external environment into the system. In this way, the runner and their immediate environment is considered as a living system, subject to the Second Law of Thermodynamics and entropy.

The simulation model of the RCAS is an abstraction of the runner's WoD as presented in Figure 10.2, and therefore not all the endogenous variables from the runner are included in the simulation model. To be included in the simulation model, the environmental variables had to be a ubiquitous measure, be subject to periodic changes and with established links to long-term running performance. Only surface type and temperature were included in the simulation model: data were readily available and the link to performance or physical output is established in the literature. Excluded from the environmental variables are altitude, the slope, road obstacles, humidity, wind, pollution, air pressure, shoes, and social engagements. Although altitude is ubiquitous, data for altitude is not reliable and the lifestyle runners tend to remain in the same geographical area for training. The slope of the road was excluded because of unreliability of the source data.

The biomechanical and physiological variables from the structural and dynamic WoD (Figure 10.2 from § 10.1 *Describing emergent running performance from interactions using the ice-berg model*) included for the simulation model were the HR, cadence, leg stiffness, VO, surface, and temperature. Data for these variables were readily available from the RW, a weather data bank (*MeteoBlue*), or found in published academic literature.

11.2.2 Parameters and dimensional consistency

There is no dimensional inconsistency of the variables or any corrective factors applied to adjust the output. Endogenous input parameters are measures used to assess running biomechanics in the real world: cadence in steps per second, VO in *meters*, leg stiffness in *kN per meter*, and have been well documented in the literature review, § 3.1.2 *The running gait*. See § 10.3.10 *Simulation input parameters and variables* for the units of variables. The basic input function to generate the training impulse (Equation 10.7), originates from well-established mathematical modelling of spring-mass systems (§ 5.1.2 *Modelling the spring-mass system with simple harmonic motion*). The concept of entropy to represent a state of order and disorder in the athlete's body, is argued from the literature and based on evolving entropy of living, open systems (see § 5.2.4 *Entropy and the athlete*).

11.2.3 Capability to solve the problem

The usefulness (purpose) and shortcomings of a model, selected time horizon, and subsequent level of aggregation are factors that impact the degree to which a systemic problem is addressed (Sterman, 2000). The purpose of the simulation model is not to solve a particular systemic problem in sport by means of analytical inspection, but rather to study hypothesised behaviour and remain open for experimentation. The outcomes from the simulation model were discussed with a pragmatic approach in § 12.5 *Discussion*. Consider therefore the simulation as a working model, subject to continued change in the future.

The inherent challenges of the hybrid simulation structure became apparent, especially harmonising the flow of time. The simulation consists of discrete events, which are managed by statechart transitions and updating functions in the agent-based component, whilst maintaining a constant flow of time in the SD component. The unit of time had to be considered carefully: the run time is per day, although calculations of training loads are in seconds and recovery and repair time in hours. All values were transformed to a portion of the day (1 hour = $^1/_{24}$ = 0.0417).

Usefulness and purpose

The simulation model is a generic version of the training cycle and does not aim to model any specific injury *per se*, but represents general patterns of recurring behaviour that lead to injury or healthy outcomes. The simulation model should relate the behaviour of the model to its system's properties (Starr, 1980). The simulation model mapped the qualitative rCLD of athletic behaviour to a quantified, dynamic version that can be adapted as the athlete follows different strategies to injury management or prevention and observe the outcome. The decisions made by the athlete are simple, yet has profound implications over time; their actions reflect the concept of dynamic complexity (§ 6.1 *Definition of systems thinking*).

The ST framework had to provide a practical departure point for sport science to study the ALS quantitatively. Articulating the RCAS as a pressurised plumbing system in the hybrid SD model facilitated the journey from linear, cause-effect thinking to causal, closed-loop thinking. The simulation model incorporated all the characteristics of complex sport systems as described by Bittencourt et al. (2016):

- The RCAS is an *open system*, interacting with the environment. The biomechanical variables are altered per different surface encountered and changes in recovery are linked with the environmental temperature.
- The RCAS is subject to *non-linearity and recursive feedback*: the system moves through critical values (or pressure points), during which the elements of the system alters their

interaction.

- The elements of the RCAS undergo *self-organisation* when the system elements interact to regain order (reversing out of the damage zones) or maintain order (conditioning, repair and recovery). This is the protective profile. When order cannot be regained, disorder of the system causes a collapse of structure and subsequent injury. This is the risk profile.

- The values of many of the parameters of the simulated RCAS were sampled from probability distributions to account for the *uncertainty* in the system.

Level of aggregation

The simulation of structure formation and damage in response to training is abstract and dimensionless. The stock variable, *structure*, absorbs all of the MSS functional fibres (bone, muscles, tendons, ligaments) into a single unit, since they exhibit the same dynamic behaviour with regard to accumulated overuse and eventual failure. This type of aggregation is in accordance with Forrester's general guidelines on aggregation (Legasto and Maciariello, 1980), see § 6.4.2 *The stock-and-flow simulation model*.

Training load as a stock variable absorbed, through SHM, the training parameters of cadence, VO, leg stiffness, and duration of training into the impulse measured in $Newton - seconds$. The level of aggregation is considered appropriate and in line with the purpose of the simulation model: to study the training behaviour that led to injury, whereby the site of the injury is not of importance.

Time horizon

The simulation model endures up to the time of injury, or for a period of 720 days injury-free. This time horizon maps accurately to the real world as between 360 to 1000 hours of running. An injury incapacitates the athlete and training is suspended, reminiscent of real-world scenarios.

System improvement

Ultimately, improvements of the system are the true test for SD models to identify the policies that lead to improvements (Forrester and Senge, 1980). The four interventions in § 12.2 *Portfolio of interventions* provided disparate changes in the behaviour of structure. The RCAS was superiorly improved when fundamental interventions were followed, as oppose to reacting to damage.

11.3 Demonstration

Demonstration of the simulation model is measured in its running costs (time and computational intensity), the integration error, a sensitivity analysis, and how the simulated output matches the real world input data.

11.3.1 Simulated output and real-world input

Table 11.1 contains the original PMF for the type of surface, together with the simulated probabilities and the error. Error margins are below 0.02 for all the surface types. The simulated output corresponds well to the original PMF.

Table 11.1: Simulation error from probability mass function for surface type input

Surface	**Simulation output probability**	**Original PMF**	**Error**
Road race	0.006	0.003	-0.003
Road running	0.417	0.399	-0.018
Trail race	0.006	0.006	0.000
Trail running	0.142	0.154	0.012
Track running	0.431	0.438	0.008

Table 11.2: Integration elasticity across time steps

Time step n	N_n **: Structure**	N_n **: Training load**
0.0005	3.362×10^{-5}	-1.79×10^{-12}
0.002	0.00597	1.26×10^{-12}
0.01	0.0036	0.0119
0.1	0.0036	0.0119

11.3.2 Integration error

A time step of 0.001 was used in the simulation model. Very small differences in integration of the most important stocks were observed for both double and half of the selected time step. A clear variation in stock levels were observed for time steps of hundredths (0.01) versus thousands (0.001). The integration measures the change in final stock for structure and training load for changes in time steps n from the base line i with a reference time step of 0.001, after 100 simulation days:

$$N_n = \frac{\texttt{stock}_i - \texttt{stock}_n}{\texttt{stock}_i} \tag{11.1}$$

Table 11.2 contains the integration measures for selected time steps. The differences in stock levels for halving the time step is negligible. Doubling the time step results in a near 0.6% change in the stock for structure. A 10-fold increase in the time step results in a 0.36% change in stock for structure, a smaller change than seen for doubling the time step. No incremental difference is seen between increasing the time step 10-fold and 100-fold. The integration of stocks seems to be stable irrespective of the size of the time step.

11.3.3 Sensitivity analysis and extreme conditions

The simulation model's robustness to changing parameters was tested under extreme conditions and by means of a sensitivity analysis. The extreme conditions test is useful for two main reasons: to discover flaws in the model's structure as well as to analyse behaviour outside of historical areas of behaviour (Forrester and Senge, 1980). The behaviour of the model was sensible when the input values were altered one by one. The sensitivity analysis assisted in finding weaknesses in the model. For instance, if the starting value of structure was too low, the model would start returning negative stock values, which is not physiologically possible.

The model was tested for its sensitivity to the initial conditions for structure (`stkStructure`), adjustments in the training pulse (increasing `flowTraining` through manipulation of the training duration), damage control and conditioning rate. Results from the sensitivity analysis are displayed in Figure 11.1 and Figure 11.2. The baseline input parameters are as follows:

1. The starting structure level (`stkStructure`) to accept the first training impulse may be thought of as a readiness measure. Readiness is set at 120% of the first training pulse (`flowTraining`): 2946850.069

2. Damage control: 4000 fibres/day (`pDamageControlZone1` in damage zone 1); 5000 fibres/day (`pDamageControlZone2` in damage zone 2)

3. Conditioning (`flowConditioning`) : 0

4. Training duration (`nTrainingDuration`): 1 hour, or 0.0417 days.

5. Recovery interval: 1 day (though not 24 hours, but 23 hours elapse from the end of the previous session to the start of the next run session).

Baseline outcome measures were observed as:

1. Time-to-injury: 53 days.

2. Fraction of the fitness gap closed: 0.13.

3. Time when fitness gap closed: did not close the gap.

4. Cumulative time in damage zone 1: 18.8 days; damage zone 2: 10 days

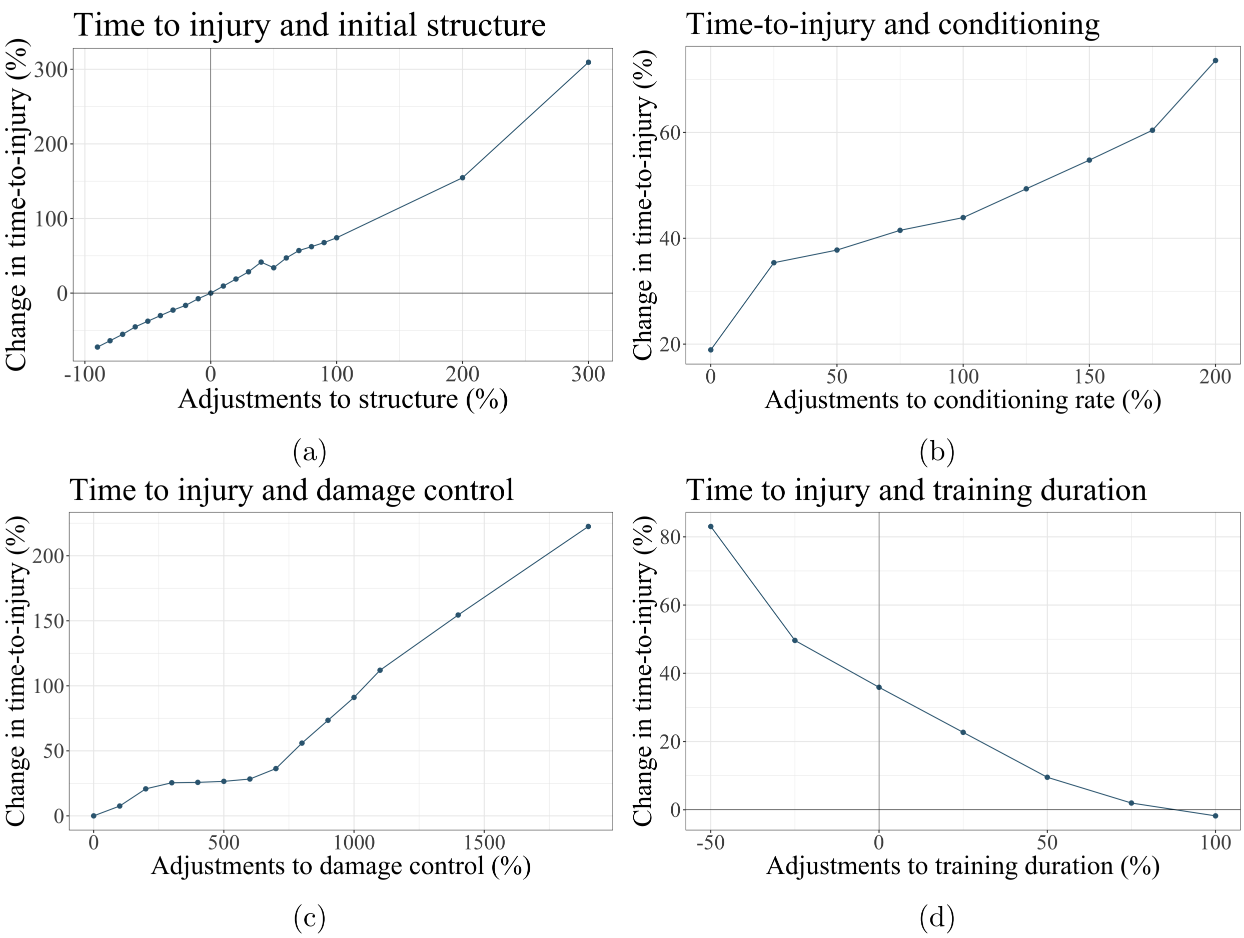


Figure 11.1: Sensitivity analysis: Time-to-injury across four adjustments

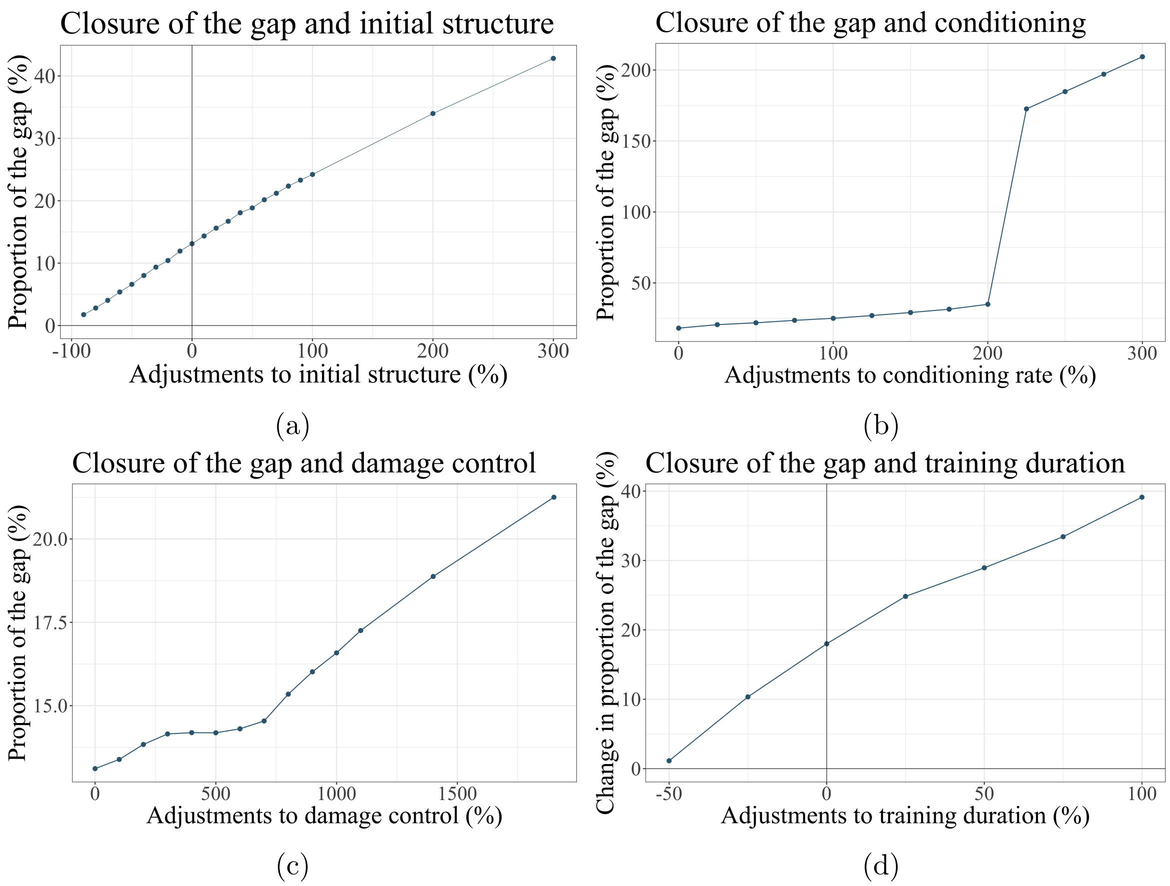


Figure 11.2: Sensitivity analysis: fitness gap closure across the four adjustments

An extreme test was performed on the initial structure (starting at 10% readiness) to calibrate the demarcation limits for the damage zones. During this test, the athlete almost immediately entered damage zone 1 after model start-up, quickly progressed to damage zone 2 and was injured on day 15. This early onset of injury is sensible, since the athlete started with poor structural integrity to receive the first training pulse and could not build enough structure in the presence of damaged structure to develop resilience.

Initial structure

The readiness measure is expressed as a fraction of the first training impulse, and ranged from the extreme of 0.1 (10%) to four times (300%) the first training pulse. The time-to-injury and initial structure follow an expected upward trajectory: higher starting values of structure takes longer to break down to the point of injury (Figure 11.1a). A 72% decrease in time-to-injury is seen for a 90% reduction in readiness, whereas a 309% increase in time-to-injury is seen for a 300% increase in readiness.

The closure of the fitness gap follows the expected positive slope in Figure 11.2a. There is a 87% decrease in the closure of the fitness gap (from base case 13.1% to 1.75% closure) for a 90% reduction in readiness, but a 159% increase in the closure of the gap for a 200% increase in structure. In the absence of additional leverage through conditioning, a high initial structure is not protective against injury.

Conditioning

At the nominal conditioning rate, there is a 19% increase in time-to-injury, when compared to the base case of no conditioning. Between 25% and 200% increase in conditioning rate the time-to-injury is gradually extended. A 200% increase in the nominal conditioning rate resulted in a 74% increase in the time-to-injury (Figure 11.1b). This change is indicative that the conditioning flow is protective against injury. The conditioning rate had a significant effect on the closure of the gap, with slow increases of the closure of the gap for conditioning rates up to 200% of the nominal. However, at a 225% increase in conditioning (3.25x the nominal rate), the athlete did not enter the damage zone at all and closed the fitness gap on day 328 without incurring an injury (Figure 11.2b). This event is marked by the step increase in closure of the gap, reaching 209% closure of the gap for the 300% increase in conditioning.

Damage control

The nominal rates of damage control were set at removal of 4000 and 5000 damaged fibres per day for damage zones 1 and 2 respectively. The changes in time-to-injury seem to stabilise between 200% and 600% increases in damage control. Only starting at a 700% increase in damage control were disparate changes observed in the time-to-injury outcome (Figure 11.1c). At 1000% increase in damage control, time-to-injury is extended by 91%. The damage control had little effect on the changes in the proportion of the fitness gap that was closed (Figure 11.2c). At 1000% increase in damage control, the closed fitness gap increased by 26.5% (still at a proportion of 16.5%).

Training duration

Figure 11.1d follows a downward curve as the training duration is increased in increments of 25% from the baseline of one hour (0.0417 days). That is, the time-to-injury increases when training duration is lowered, but decreases when training duration is increased. At a 50% reduction in training duration, there is a 35% increase in the time-to-injury. At 100% increase in training duration, the time-to-injury has decreased by 28%.

11.3.4 Equilibrium and disequilibrium

The model exhibited both disequilibrium and equilibrium behaviour, which depended on the decisions of the athlete on how to approach their training. Equilibrium was achieved when delays were respected and training loads did not overwhelm the structure, whilst disequilibrium was characterised by eroding structure from too frequent training and higher training loads beyond the repair-and-renew capacity. This type of behaviour was expected.

11.3.5 Family member tests

The simulation model was tested in § 12.4 *Same simulation model, (somewhat) dissimilar systems* with input data from different athletes. The different athletes are essentially representing family members of the same system, or different versions of the RCAS. Dissimilar outcomes were observed in these tests, which were expected. The magnitude of some events differed, although the general behaviour of the system remained similar.

11.3.6 Costs of running the model

The efficiency of algorithms may be measured in time and space measures (Mohammed et al., 2016). The simulation model was built using the Personal Learning Edition 8.5.1 of the Anylogic software, making it accessible to any personal computer with the minimum space and computational memory requirements for the software. Costs to run the model are minimal. It takes 830 seconds to complete 720 days in the simulation model in normal mode at between 0.8

and 1 simulation days per second, completing 305636 model steps and uses between 6% and 19% of the available 512M memory. In virtual mode, it takes 117 seconds to run 720 days at between 5.1 and 7.8 days per second, and uses between 4% and 6% of the available memory. Microsoft Excel is required to capture the output data for comparisons between parameter settings.

11.4 Conclusion

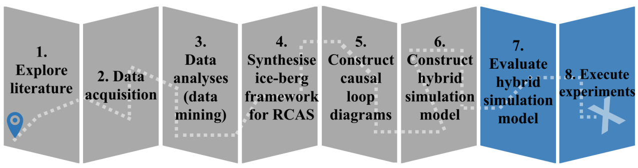
The ST framework made a research contribution to the knowledge fields in both theory and pragmatism. Despite not having a real-world counterpart, the simulation artefact maps accurately to the real world as a conceptual model; a novel way of thinking about fitness and injury. The simulation model was shown to be practical in its capability to find optimal leverage for system improvements. The simulation's output is representative of the input data and achieves this with low running costs.

Chapter 12

Results from the runner as a complex adaptive system

Contents

This chapter formulates part of task 7 and the entire task 8 from the research methodology.



The RCAS was presented in Chapter 10 as a hybrid agent-based, SD model. Model evaluation was completed in Chapter 11. This chapter presents the results from the simulation model, with various intervention strategies to find optimal leverage.

12.1 Interventions to find leverage

The experimentation process considered four factors as policy levers in the training cycle, namely the conditioning rate (`flowConditioning` as no conditioning versus some multiple of the nominal conditioning rate), damage control rate (through `pDamageControl`), more training in response to poor fitness, and the recovery interval (one or two days). Two of these levers were adjusted one at a time during the sensitivity analysis (conditioning and damage control) earlier in § 11.3.3. Combinations of the levers made up the portfolios of interventions (see Table 12.1). Adjustments are organised as follows:

1. A factor adjusting the conditioning rate (`flowConditioning`) between 'off' (no conditioning) and 'on' at the nominal rate, then adjusted from 1.25 to four times the nominal rate, in increments of 0.25.

Table 12.1: Interventions tested

Intervention type	Damage control	More training	Conditioning	Recovery interval	Sensitivity analyses
Quick fix	X				Yes
Fundamental			X		Yes
Quick fix	X	X			No
Mix	X		X		No
Mix	X		X	X	No

2. Damage control (`pDamageControl`) adjusted upwards to 20 times the nominal rate within the damage zones.

3. A switch (`pIncrTrainingDurationSwitch`) to activate extra running training by adding time to the training duration, `nTrainingDuration`.

4. The recovery interval (timing between successive training sessions) is altered between baseline of one day and the alternative of two days.

Outcomes that were considered are the time-to-injury (if it did occur), the closed fraction of the fitness gap, the time when the gap was closed (if it did occur) and the cumulative time spent in the damage zones.

12.2 Portfolio of interventions

A portfolio of interventions consisting of conditioning, damage control, and the recovery interval were modelled. Four general outcomes were observed from the simulation's portfolios:

1. Outcome 1: the athlete sustained an injury.

2. Outcome 2: the athlete was injury-free, but could not close the gap and spent time in damage zones.

3. Outcome 3: the athlete remained injury-free, closed the gap, but did spend time in the damage zones.

4. Outcome 4: the athlete remained injury-free, closed the gap, and did not spend any time in the damage zones.

The results from the simulations are grouped according to the interventions and the outcomes. Tables 12.2 to 12.4 contain the listed outcomes.

12.2.1 Outcome 1: injuries sustained

In Table 12.2, the majority of injuries occurred under the 1-day recovery interval, with some injuries occurring under the 2-day recovery interval when there was no conditioning. The shortest time-to-injury is for the base case at 53 days, followed by the quick fix combination of some damage control and more training at 54 days. The longest time-to-injury is for a 2-day recovery interval on 9x increase in damage control and no conditioning, at 222.7 days. This event indicates that despite a strong and intense damage control reaction and a fundamental approach to recovery, the accumulation of damage was still severe enough over time to build up to an injury.

Table 12.2: Outcome 1: injuries sustained

Portfolio (adjustments from baseline)	**Gap closed (%)**	**Time-to-injury (days)**	**Time in damage zone 1 (days)**	**Time in damage zone 2 (days)**
base case: initial structure = training pulse	13.11	53.01	18.81	10.00
3x ↑ dc; ∼ cnd; 1-day rcv	19.21	70.66	42.00	0.00
3x ↑ dc; 1.25x ↑ cnd; 1-day rcv	20.65	71.94	42.00	0.00
3x ↑ dc; 1.5x ↑ cnd; 1-day rcv	21.95	73.23	42.00	0.00
3x ↑ dc; 1.75x ↑ cnd; 1-day rcv	23.63	75.01	42.00	0.00
8x ↑ dc; ∼ cnd; 1-day rcv	22.40	94.27	42.00	0.00
8x ↑ dc; 1.25x ↑ cnd; 1-day rcv	25.49	105.08	42.00	0.00
8x ↑ dc; 1.5x ↑ cnd; 1-day rcv	29.84	119.89	42.00	0.00
9x ↑ dc; ∼ cnd; 1-day rcv	24.63	112.34	42.00	0.00
9x ↑ dc; 1.25x ↑ cnd; 1-day rcv	27.91	121.42	42.00	0.00
9x ↑ dc; 1.5x ↑ cnd; 1-day rcv	33.29	141.20	42.00	0.00
10x ↑ dc; ∼ cnd; 1-day rcv	25.98	122.52	42.00	0.00
10x ↑ dc; 1.25x ↑ cnd; 1-day rcv	30.54	139.04	42.00	0.00
10x ↑ dc; 1.5x ↑ cnd; 1-day rcv	37.66	169.12	42.00	0.00
3x ↑ dc; more training; 1-day rcv	13.56	54.05	19.81	10.00
8x ↑ dc; more training; 1-day rcv	15.18	67.87	42.00	0.00
9x ↑ dc; more training; 1-day rcv	15.23	68.85	42.00	0.00
10x ↑ dc; more training; 1-day rcv	15.41	71.55	42.00	0.00
3x ↑ dc; no cnd; 2-day rcv	12.56	92.26	42.00	0.00
8x ↑ dc; no cnd; 2-day rcv	15.42	198.61	42.00	0.00
9x ↑ dc; no cnd; 2-day rcv	15.96	222.70	42.00	0.00

dc - damage control; cnd - conditioning; rcv - recovery interval; ↑ - upward adjustment; ∼ - no change from nominal

12.2.2 Outcome 2: injury-free, but did not close the fitness gap

Other portfolios in Table 12.3 did protect against injury, but the athlete sustained some damage to structures. The injury-free group consists of two types of portfolios for a 2-day recovery interval: increasing conditioning whilst damage control remains constant, and increasing damage control whilst conditioning remains constant. Keeping conditioning constant at the nominal rate and only increasing the damage control resulted in a gap closure of 63%, but less time spent in the damage zone 1 for increasing damage control. Gap closures of 96% are observed for conditioning rates of 1.75x the nominal, regardless of damage control. The gap-closure figures repeat for each increasing level of damage control and paired conditioning rate. This is because the athlete never entered the damage zone 1 when exceeding the nominal conditioning rate and the damage control action never had to be taken. Damage control therefore had no effect on the level of structure reached if the athlete did not enter the damage zone, which is in accordance with the flow of the model explained in § 10.3.6 *Progressing through the damage zones*. The $^d/_s$-ratio remained below the lower threshold for these interventions. Only small positive differences (1.5%) in the closure of the fitness gap are seen for 10% increases in the training pulse.

12.2.3 Outcomes 3 and 4: injury-free and closed the fitness gap

The portfolio's which closed the fitness gap (Table 12.4) started at 1.75x the nominal conditioning rate with damage control remaining constant for a 1-day recovery interval; conditioning had to be 2x the nominal rate for a 2-day recovery interval to close the gap.

Table 12.3: Outcome 2: injury-free, but could not close the gap

Portfolio (adjustments from baseline)	Gap closed (%)	Time in damage zone 1 (days)	Time in damage zone 2 (days)
3x ↑ dc; ∼ cnd; 2-day rcv	62.96	18.24	0.00
3x ↑ dc; 1.25x ↑ cnd; 2-day rcv	73.06	0.00	0.00
3x ↑ dc; 1.5x ↑ cnd; 2-day rcv	84.52	0.00	0.00
3x ↑ dc; 1.75x ↑ cnd; 2-day rcv	95.97	0.00	0.00
8x ↑ dc; ∼ cnd; 2-day rcv	63.00	3.25	0.00
8x ↑ dc; 1.25x ↑ cnd; 2-day rcv	73.06	0.00	0.00
8x ↑ dc; 1.5x ↑ cnd; 2-day rcv	84.52	0.00	0.00
8x ↑ dc; 1.75x ↑ cnd; 2-day rcv	95.97	0.00	0.00
9x ↑ dc; ∼ cnd; 2-day rcv	63.00	2.79	0.00
9x ↑ dc; 1.25x ↑ cnd; 2-day rcv	73.06	0.00	0.00
9↑ dc; 1.5↑ cnd; 2-day rcv	84.52	0.00	0.00
9↑ dc; 1.75↑ cnd; 2-day rcv	95.97	0.00	0.00
3x ↑ dc; ∼ cnd; 2-day rcv; 10 tp	64.47	19.48	0.00
3x ↑ dc; 1.25x ↑ cnd; 2-day rcv; 10 tp	74.48	0.00	0.00
3x ↑ dc; 1.5x ↑ cnd; 2-day rcv; 10 tp	86.07	0.00	0.00

dc - damage control; cnd - conditioning; tp - training pulse; ↑ - upward adjustment;
∼ - no change from nominal

Table 12.4: Outcomes 3 and 4: injury free and closed the gap

Portfolio (adjustments from baseline)	Gap closed (%)	Time to close the gap (days)	Time in damage zone 1 (days)
8x ↑ dc; 1.75x ↑ cnd	103.70	663.00	35.18
8x ↑ dc; 2x ↑ cnd	114.22	562.00	24.40
8x ↑ dc; 2.25x ↑ cnd	125.34	490.00	16.01
8x ↑ dc; 2.5x ↑ cnd	136.84	436.00	9.27
8x ↑ dc; 2.75x ↑ cnd	148.60	392.00	5.48
8x ↑ dc; 3x ↑ cnd	160.54	357.00	2.04
8x ↑ dc; 3.25x ↑ cnd	172.62	328.00	0.00
9x ↑ dc; 1.75x ↑ cnd	103.71	663.00	28.77
9x ↑ dc; 2x ↑ cnd	114.24	562.00	20.07
9x ↑ dc; 2.25x ↑ cnd	125.35	490.00	13.25
9x ↑ dc; 2.5x ↑ cnd	136.84	435.00	7.76
9x ↑ dc; 2.75x ↑ cnd	148.60	392.00	4.60
9x ↑ dc; 3x ↑ cnd	160.54	357.00	1.72
9x ↑ dc; 3.25x ↑ cnd	172.62	328.00	0.00
10x ↑ dc; 1.75x ↑ cnd	103.73	663.00	24.31
10x ↑ dc; 2x ↑ cnd	114.24	562.00	17.08
10x ↑ dc; 2.25x ↑ cnd	125.35	490.00	11.32
10x ↑ dc; 2.5x ↑ cnd	136.85	435.00	6.69
10x ↑ dc; 2.75x ↑ cnd	148.61	392.00	3.97
10x ↑ dc; 3x ↑ cnd	160.54	357.00	1.49
10x ↑ dc; 3.25x ↑ cnd	172.62	328.00	0.00
3x ↑ dc; 2x ↑ cnd; 2-day rcv	107.37	639.00	0.00

dc - damage control; cnd - conditioning; rcv - recovery interval; ↑ - upward adjustment

12.2.4 Behaviour of structure across portfolios

There is a marked difference in structure formation behaviour between a 1-day and a 2-day recovery interval: for 1-day, there is a step increase in closure of the fitness gap. Figure 12.1 graphically depict the results per outcome (injured or injury-free) with adjusted conditioning rate and recovery interval across all damage controls. To remain damage-free (that is, no time spent in the damage zones) the athlete must complete a minimum of 3.25x the nominal conditioning rate for a recovery interval of one day, compared to 1.25x the nominal conditioning rate for a 2-day recovery interval. To reach their goal, an athlete must maintain a minimum of 1.75x nominal conditioning rate for a 1-day recovery interval and 2x nominal conditioning for a 2-day recovery interval. Under the 2-day recovery interval, the athlete only got injured for the case of 0 conditioning, whereas for the 1-day interval the athlete got injured up to a 1.5x nominal conditioning rate.

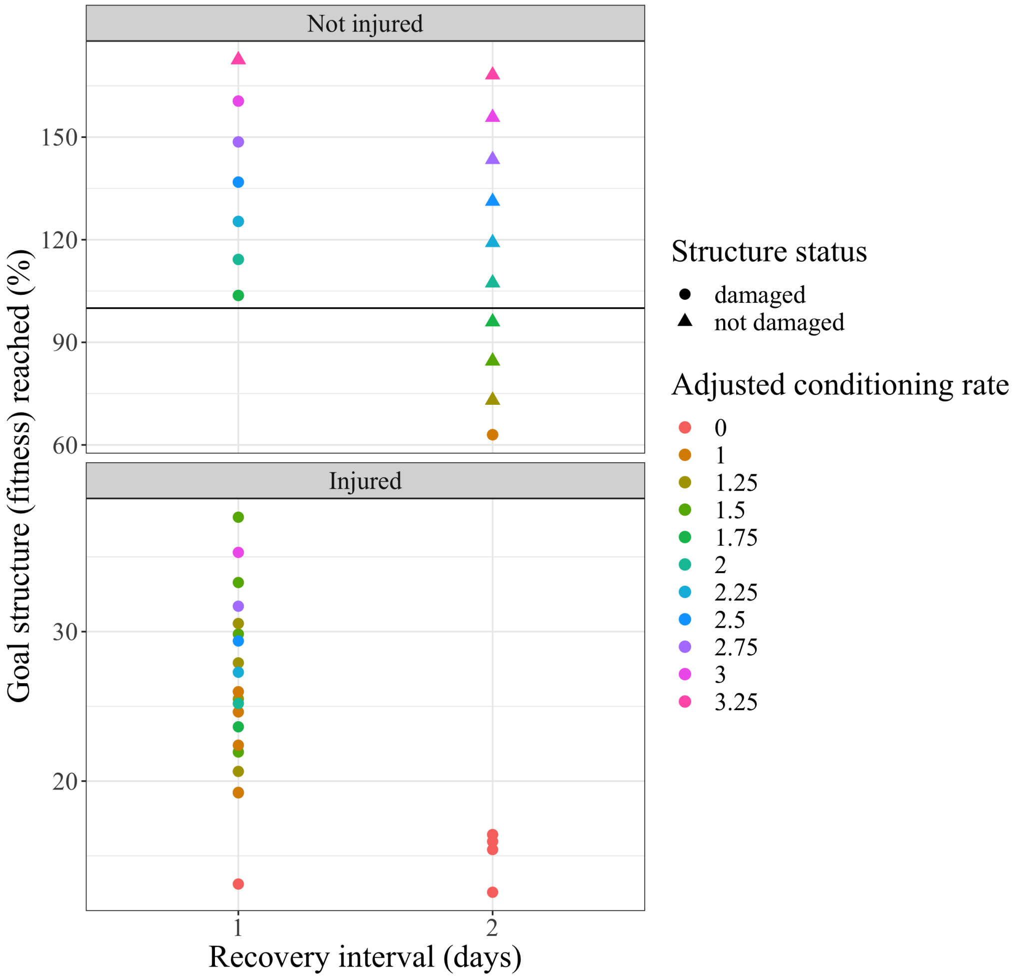


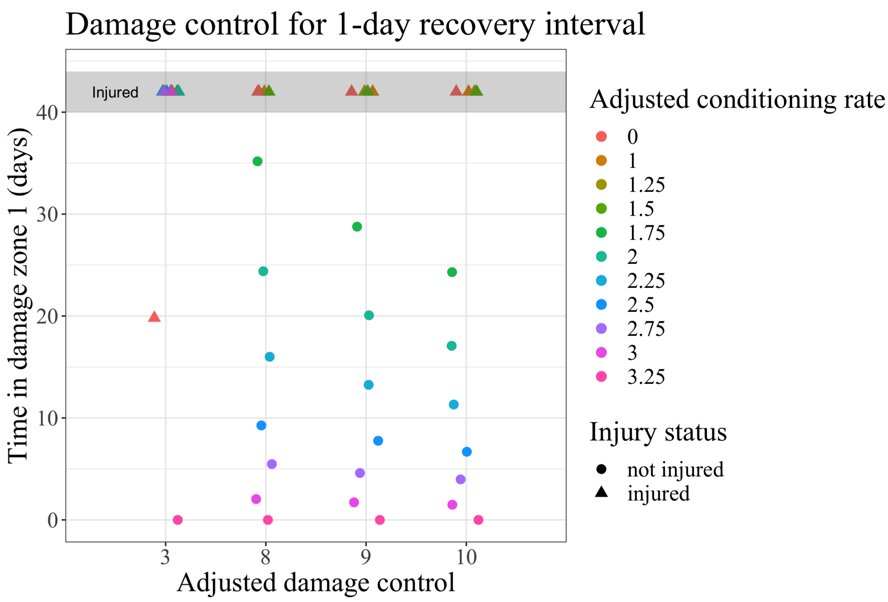
Figure 12.1: Outcomes across recovery intervals

Damage control is shown in jitter plots in Figures 12.2a and 12.2b for levels where incremental changes in time-to-injury were observed from the sensitivity analysis (Figure 11.1c), namely at 3x and 8x – 10x the nominal damage control rates. The behaviour across the injured and not-injured outcomes is different across the damage zones. For injured outcomes, there are disparities between the level of structure reached across the damage controls and conditioning rates. Higher damage control and higher conditioning rates resulted in a higher percentage closure of the gap. However, there are no changes in the proportion of the fitness gap closed when the athlete was not injured, and had reached their goal across the damage controls. Conditioning drives structure formation in the uninjured group, not damage control.

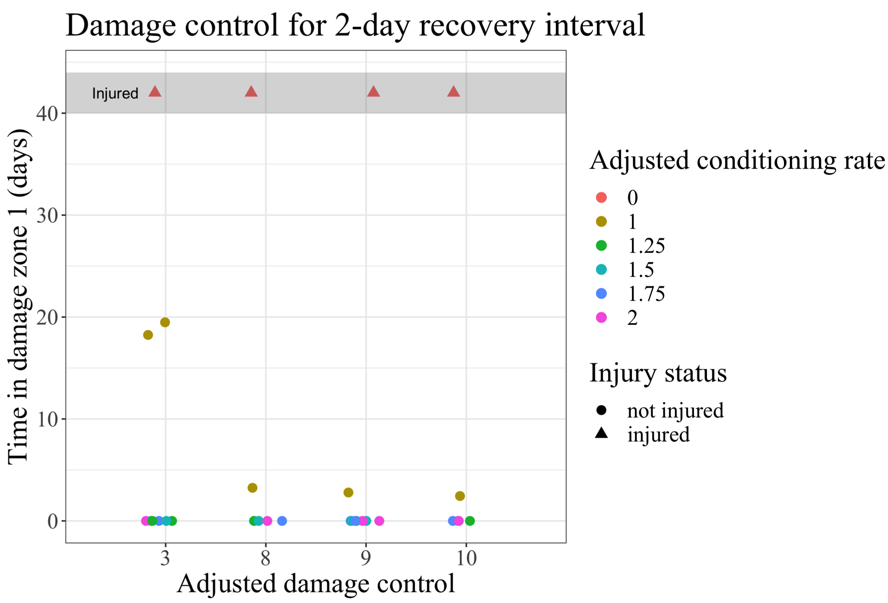
The rate at which the fitness gap is closed differs for the recovery intervals and across the adjustments of the conditioning rates, as expected. The time to close the fitness gap decreases for increasing conditioning rates. The gap is closed starting at a conditioning rate of 1.75x the nominal for a 1-day recovery interval and 2x the nominal for a 2-day recovery interval. The 1-day recovery is quicker to close the gap than the 2-day recovery, having had a higher cumulative training load by training each day.

12.3 Gauging fitness and health: the damage to structure ratio

The $^{d}/_{s}$-ratio determines the intensity scales of the damage control and conditioning rates of the athlete, and is in turn also influenced by such changes. The ranging $^{d}/_{s}$-ratios output from the interaction with conditioning adjustments (per constant damage control) for the two recovery intervals are shown in Figure 12.4. The $^{d}/_{s}$-ratio behaves non-linearly for all the adjustments. First rising sharply, then displaying some stability around 0.09 (the lower limit for the first damage zone) followed by exponential decline, except for the instances where an injury occurred and the simulation had stopped. The rate of decline is slower for lower conditioning rates, seen for the curves with less steep slopes in Figure 12.4. Lower conditioning rates result in less structure formation, and a higher $^{d}/_{s}$-ratio. The stable behaviour surrounding a $^{d}/_{s}$-ratio of 0.09 results from the damage control adjustments made in the model when the athlete enters the damage zone. During this time, the athlete remains close to the limit or within the damage zone, before enough structure is built for the $^{d}/_{s}$-ratio to start its decline. The athlete never enters the damage zone for a 3.25x and 1.25x nominal conditioning rate for the 1-day and 2-day recovery intervals respectively, and the curve immediately goes into decline after reaching its peak (0.089 for both recovery intervals). The lowest peak $^{d}/_{s}$-ratio is obtained for a 2-day recovery interval and 3.25x conditioning rate, at 0.047.



(a)



(b)

Figure 12.2: Outcomes from damage control

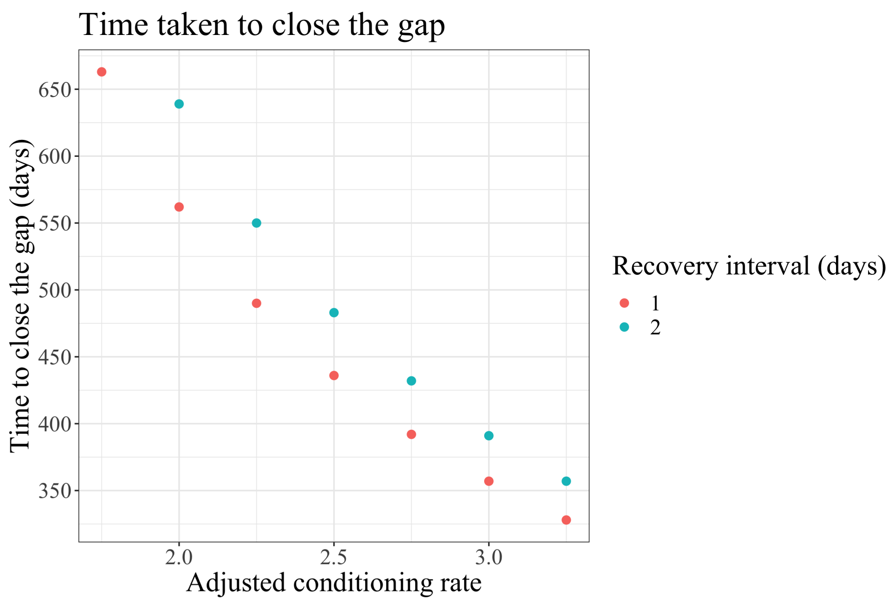


Figure 12.3: Closure of the fitness gap

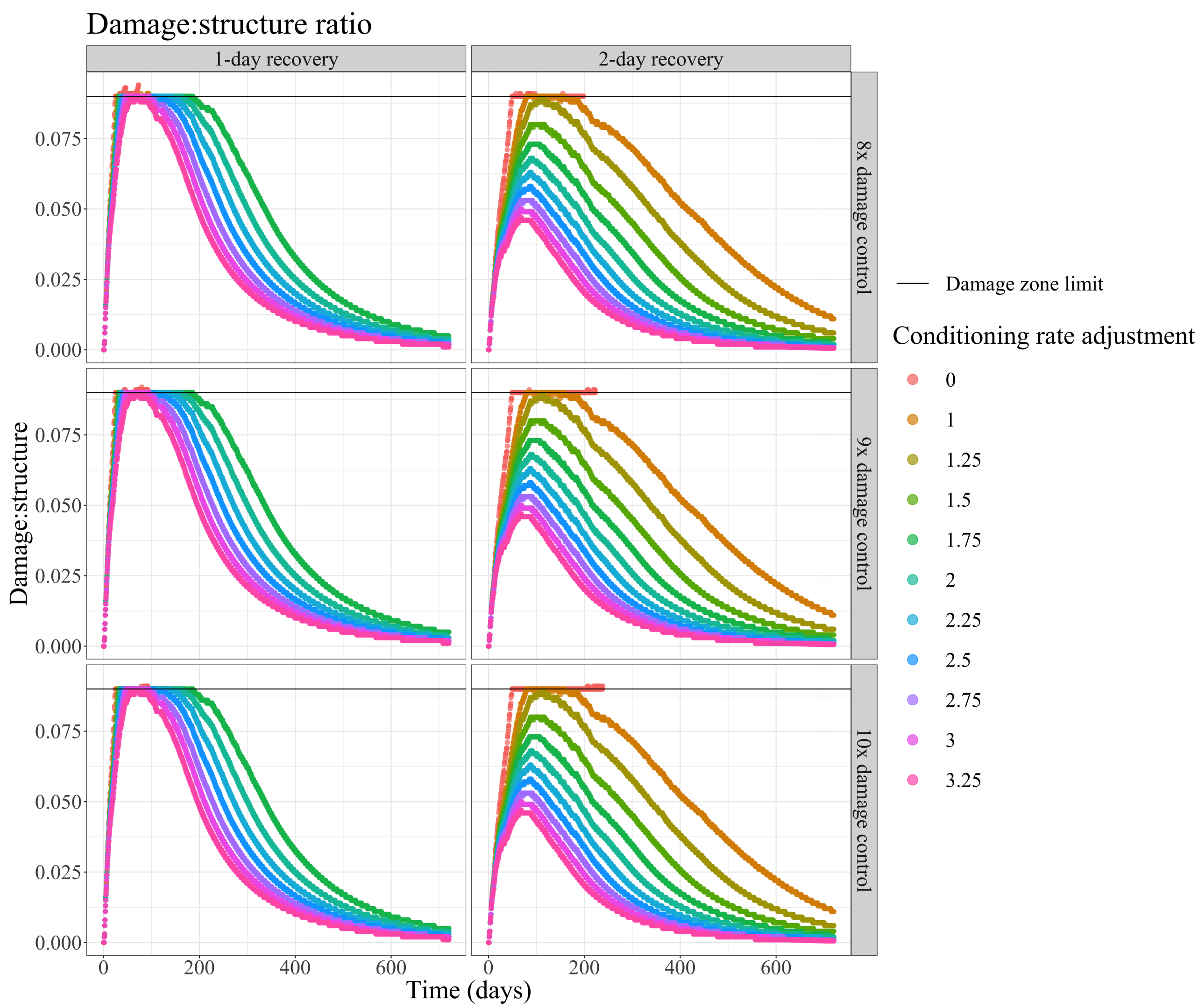


Figure 12.4: The damage:structure ratio for augmentations in the conditioning rate

12.4 Same simulation model, (somewhat) dissimilar systems

The simulation model was tested for behaviour anomalies by substituting values of the input parameters with values from other athletes' RWs, henceforth referred to as 'athlete 2' and 'athlete 4'. Their input parameters may be found in Appendix A. The simulation was run for the base case (1x damage control, 0 conditioning) and for increasing conditioning rates on a 8x damage control level for the two additional athletes.

Some disparities are visible in the time-to-injury for the athletes (Figure 12.5). This is indicative that the systems' behaviour is different in magnitude of outcome metrics, but the patterns are similar. Athlete 3 consistently takes longer than athlete 2 to reach an injury, however athlete 3 does not get injured for a conditioning rate of 1.75x whereas athlete 2 does get injured. Athlete 4 is consistently injured earlier than the other two.

Figure 12.6 displays in a jitter plot the time spent in the damage zones by the athletes. Athlete 2 spends more time per conditioning level in the damage zones than athlete 3, and only moves completely away from the damage zone on a conditioning rate of 3.5x the nominal rate. Athlete 4 is only sheltered from time in the damage zone by a 3.75x conditioning rate.

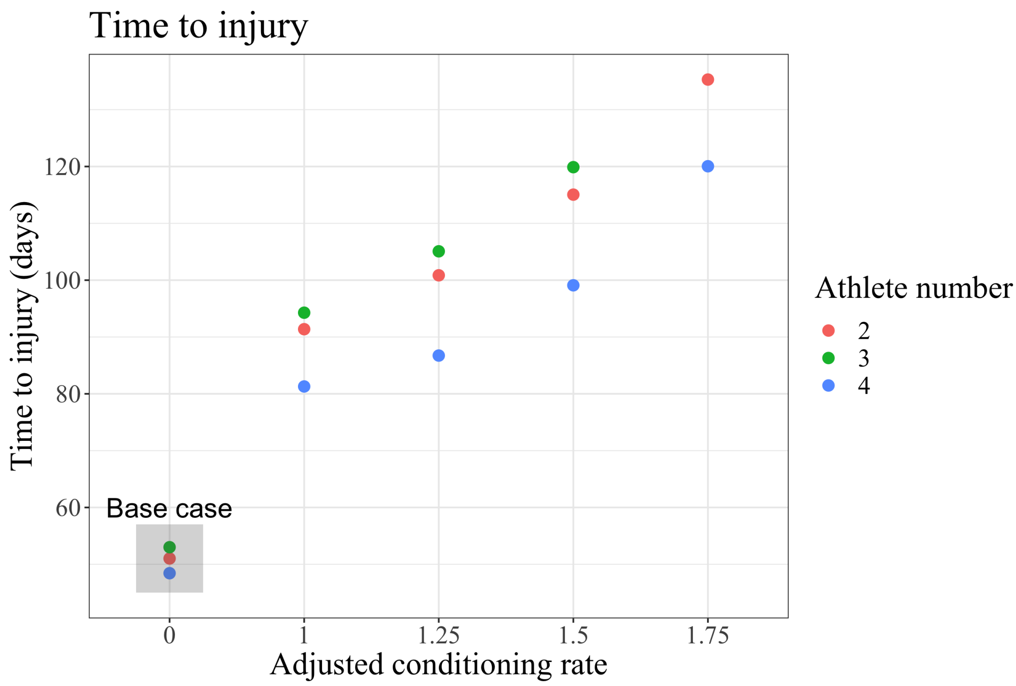


Figure 12.5: Time to injury for athletes 2 to 4

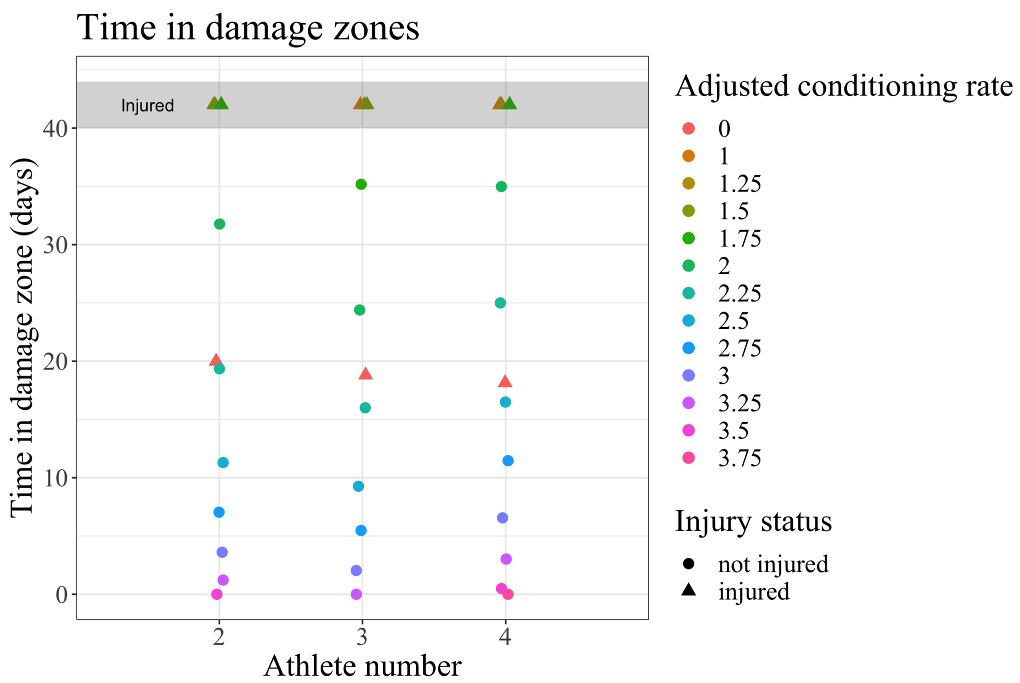


Figure 12.6: Time in damage zone for athletes 2 to 4

Figure 12.7 shows the percentage of the gap that was closed for athletes 2 to 4. The same levels of gap closures are seen for conditioning adjustments. The minimum conditioning rate of 2x to remain injury-free and close the gap for athlete 2 is reflected in this graph, compared to the 1.75x conditioning rate for athlete 3. Athlete 4 closes the gap on a minimum of 2x conditioning rate, and also manages to reach the highest fitness level at 3.75x conditioning rate.

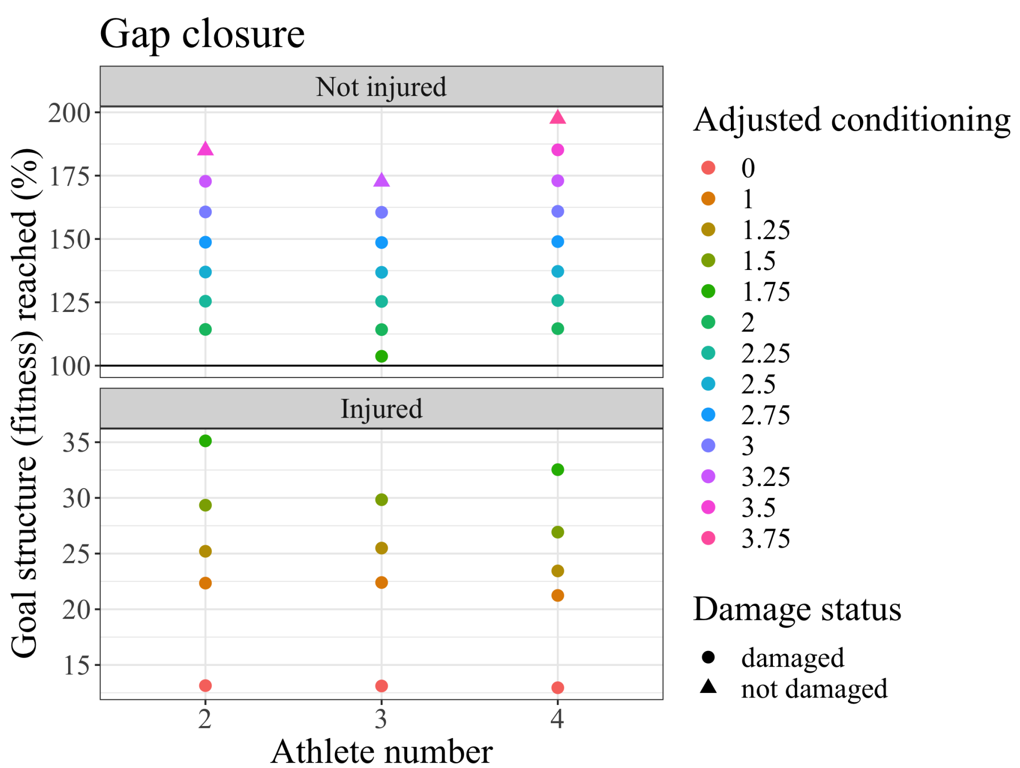


Figure 12.7: Proportion of the structure reached for athletes 2 to 4

12.5 Discussion

The simulation model formed part of the process to answer the second research sub-question of this research project, namely: *How can the ST perspectives be instantiated through computational applications?* The development and demonstration of the model should address the lack of pragmatism of ST and computational modelling in sport science. The key findings from the simulation reinforce the systemic closed-loop thinking from the ST paradigm (§ 6.1 *Definition of systems thinking*). The effects from training (damaged fibres) are causes for alterations in the training behaviour. By changing structural elements through parameter variation, based on the feedback from the state of damage or integrity of the system, the behaviour of the athlete is influenced, which results in different effects (become injured, return from the damage zones or close the fitness gap).

12.5.1 Patterns of behaviour

Multiple experiments were conducted in the simulation model with various interventions to drive the athlete's structure formation. The growth-and-underinvestment archetype system served as the basis from which the interventions were developed. The results showed that fundamental solutions, namely increasing the recovery interval and higher conditioning rates, aids the athlete in maintaining healthy structures and avoid injuries. Re-active interventions to fix the problem quickly (to get out of the damage zone) did not protect against injury.

A pattern became evident with regard to the conditioning rates. Significantly, there seems to be two pivotal conditioning rates:

1. A return-from-damage point; this is the rate at which the athlete is able to exit the damage zones and endure to remain injury-free.

2. A protective rate, the rate at which the athlete is completely prevented from entering the damage zone (because there is sufficient and sturdy structure formation, and therefore maintained a low $^d/_s$-ratio).

At the protective conditioning rate, the athlete remains free of damage and may close the fitness gap, depending on the recovery interval. The level of damage control becomes irrelevant, since the athlete never entered the damage zone. This pivotal protective conditioning rate differs between the recovery intervals. For a longer recovery interval, the body had time to return order to the disarrayed fibres before the next training bout, thereby requiring a lower conditioning rate (1.25x for a 2-day recovery interval). A shorter recovery time means that complete order had not yet been restored after the last bout before the next training session starts, therefore a higher conditioning rate to prepare enough structure to absorb the load, is required (3.25x for the 1-day recovery interval).

In conjunction with the return-from-damage conditioning rate, there is a minimum effective damage control response. This is the damage control level required to remove enough damage for the athlete to be able to move out of the damage zone. However, damage control as a stand-alone intervention proved to be ineffective against injury development. The athlete required a return-from-damage conditioning rate of 1.75x on at least 8x damage control level to return from the damage zone for a 1-day recovery interval, whereas on the 2-day recovery interval the athlete required a minimum of 3x damage control at the nominal conditioning rate.

The introduction of decision points based on the $^d/_s$ ratio altered the behaviour of the model, which Bittencourt et al. (2016) referred to as the critical values when the interacting elements of a system configure to produce these patterns or regularities. Unexpectedly, a success-to-the-successful archetype emerged from the simulation model: once within the first damage zone and an athlete opted for the quick fixes or shifted the burden, they remained in the damage zones and a vicious cycle towards injury developed, despite attempts to clear the damage. The damaged structure kept accumulating and structure diminished in size, resulting in continuing higher $^{d}/_{s}$-ratios. Otherwise, in more favourable $^{d}/_{s}$-ratios when sufficient structure was in place, the athlete only improved their fitness and damage accumulation diminished gradually in size. The athlete spent minimal time in the damage zones and progressed in a virtuous cycle; success was granted to the structural integrity with continued lowering of the $^{d}/_{s}$-ratio. This type of systemic behaviour is not obvious to an athlete who trains hard in the present time, but with lack of insight into future events emerging from current behaviour. The delays between training and structure formation challenge an athlete to maintain a balance between training and recovery.

Whether the athlete closed their fitness gap was a secondary outcome from experimentation. The time taken to close the gap decreased with increasing conditioning rates, which was expected. A 2-day recovery interval prolonged the time to close the gap on the same level of conditioning for the 1-day recovery interval, although the athlete remained mostly damage-free on the 2-day recovery interval.

12.5.2 Implications

Other recent ST frameworks and simulation models in sport focus on outcomes on higher (macro) complex system levels (McLean et al., 2019; Hulme et al., 2017b, 2019), whereas the simulation model demonstrated here is subject-specific on the individual (meso) and subsystem (organ) level with incorporation of the MSS and CVS, to dynamically and quantitatively augment understanding of the RCAS in their environment. The subject-specific ST approach was also qualitatively illustrated in the complex sport model from Bittencourt et al. (2016), showing different injury development patterns (or risk profiles) for athletes from the sports of basketball and ballet. A subject-specific, yet systemic approach, aligns with the paradigm shift in medicine and healthcare in the 21st century, often referred to as the P's of healthcare (Golubnitschaja et al., 2016): first, *personalised* activity as each athlete has unique abilities and weaknesses; second, *participation* from the individual by the honing of skills and subsequent physical performance; third, focus is placed on *prevention* of injuries; fourth, the individual must be *precise* in their training and execution to attain their specific sporting goals and be an effective athlete.

Hulme et al. (2019) also encouraged data driven computational applications. The utilisation of personal data from RWs to drive the simulation model adds to the personalisation and preciseness of the model, similar to how Kosmidis and Passfield (2015) and Ahmad et al. (2018) found subject-specific metrics in their respective analyses of data from RWs.

The results provide support for a ST approach in sport science, whereby the mental models of athletes and practitioners may be challenged both conceptually and empirically. The quantitative modulations of the causal interdependencies within the hybrid SD model helped identify optimal leverage points (or targets for interventions, as mentioned by Hulme et al. (2019)), as well as the system elements reactions to their implementation. The simulation model showed that respecting delays and opting for fundamental solutions, even though they take longer to materialise, are optimal for structural integrity and athletic longevity. These concepts and interventions are not new to the sporting world; however the method in which they are explored is novel. Thinking about training and overuse injuries in terms of a pressurised plumbing system (§ 6.4.2 *The stock-and-flow simulation model*) with accumulating content in tanks and actions as valves to increase or release content by allowing movement between the tanks, departs from a linear cause-effect thinking pattern to arrive at causal, closed-loop thinking. The site of accumulation in the system drives the development of healthy structure or injury. Intervention actions that add to the flow of structure formation (conditioning) and allow for less outflow from structure (longer recovery) result in accumulation of the structure. Actions that open the outflow from structure more than the inflow to structure (short recoveries) result in damage build-up elsewhere in the system, leading to injury. The actions that open the outflow of accumulated damage are not effective to prevent damage build-up.

The relationship between the level of structure and damage (the $^d/_s$-ratio) is considered as the main pressure point and an indicator of health or injury. The inciting of the injury was not based on published injury rates, but rather on emergent behaviour and the critical values of the relationship between structure and damage. Inciting an injury as an anticipated future event (through the simulation parameters) adds to the confidence of the SD model to study structural interactions in the RCAS.

Practical implications of the results are now considered. The daily conditioning rate is only modelled as a percentage of the initial training pulse. Conditioning should be interpreted as any activity undertaken by the athlete to advance their structure formation without or with less impact loading, by introduction of *negentropy*. This may include lifestyle changes to incorporate more movement, exercise therapy to strengthen muscles to handle the load (Barton, 2018), cross-training without impact loading (example, swimming and cycling) (Foster et al., 1995; Paquette et al., 2018), running retraining to stimulate and maintain the neuromuscluar pathways to improve running biomechanics (Barton, 2018), improved nutrition (Brukner and Khan, 2006; Kreher and Schwartz, 2012), and quality sleep (Kreher and Schwartz, 2012). An athlete wishing to run every day (that is, they have a 1-day recovery interval), must incorporate a conditioning rate higher than an athlete who utilises a longer recovery, yet both are able to close the gap. These mechanical type of decisions will place a time demand on the athlete.

Damage control is any action taken by the athlete to instantly reduce the load placed on the body or remove damaged fibres; this may be through the use of orthosis, insoles, taping, specialised footwear (Barton, 2018), regular sports massage, compression garments (Bindemann, 2012; Engel et al., 2016) and so forth. Damage control places a resource demand on the athlete to obtain the extra products or services, and considering the physical products are subject to mechanical wear, the demand is recurring.

In the real world, however, it is sensible to consider a combined training approach, with a fundamental intervention to build structure and some form of damage control to release damaged structure. Many factors may limit an athlete's capacity to invest in fundamental interventions (such as time constraints to maintain the required conditioning rate), and therefore to employ some damage control as part of the training programme is reasonable and practical.

12.5.3 Limitations

The limitations of the simulation model are related to methodological processes and physiological assumptions. In order to maintain parsimony in the model, the simulation is an abstraction of the real world and does not account for all the possible permutations that an ALS may undergo during training. The model assumes the athlete's capability to adapt, but already these adaptation rates differ amongst individual athletes and are subject to change under environmental conditions (Borresen and Lambert, 2009). The training load calculation through the pulsed functions (Equations 10.7 and 10.8) only quantifies the interaction between the MSS and the surface. Although the HR zones are utilised to gauge repair time (the full return to homeostasis), more work is required to quantify the aerobic response. Some physiological and biological variables that are excluded from the model are genetic factors and biometrics. The athlete's HRV was not directly incorporated into the model (since it is not a direct variable extracted from the container files of the RWs), however the HR zones were considered in the recovery delay instead. Other social and environmental factors were also excluded, such as:

- Psychological processes, for example the motivational drive to train.

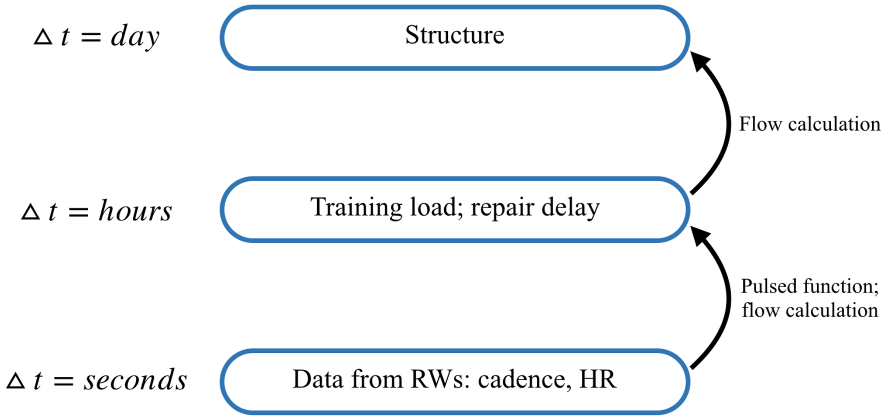


Figure 12.8: Levels of analysis

- Social-economic status of the athlete.
- Environmental factors are not exhaustive and were excluded due to practical reasons, for example traffic congestion, 'traffic furniture' such as road islands, infrastructural challenges such as potholes, weather related variables (relative humidity).
- Time and other resource constraints.

There are factors that were outside the scope of this research project. Dealing with a system with expressions on various levels of detail it was necessary to decide upon the level of unit analysis for tractability. This is similar to what Coveney et al. (2013) refer to as the scale upon which system biologists perform analysis, then moving up and down to incorporate higher and lower levels of detail. The model may be up-scaled to include the macro-level (wider sociocultural and political environments) or downscaled to include more detailed micro-level, physiological behaviour (Hulme et al., 2019). The connectedness of the varying constraints and dynamics at different spatio-temporal scales is mentioned in Balaguè et al. (2013) as an important area of research. The instantaneous changes in the body during running (captured in small time-intervals on the RWs) were aggregated into the training load or as part of the homeostatic repair delay (Figure 12.8). This type of aggregation may hide details or patterns of change during the run that may influence recovery later on.

Some injury history was initially obtained from participating athletes; however, the question regarding the detail of the injury was set up before the simulation model was constructed. During construction, it was realised that the collected data were not comprehensive enough to use as part of the process to incite an injury. Improved and more detailed data collection on injury history is required.

12.6 Conclusion

The model does not have a direct real-world counterpart, but is rather a high-level abstraction of the adaptive processes in the human body in response to training. The simulation model is considered a conceptual representation (the fitness of a runner as fluctuating integrity of the structure of body tissues) of a concrete system, namely the ALS. Newtonian time ($\triangle\ t$) is used in the calculations, although the process of entropy governs changes along the unidirectional arrow of time, T. Fundamental interventions, aimed at altering the structural level of the RCAS, are successful to build a healthy, resilient athlete. Symptomatic interventions (quick fixes) must only be used in support of the fundamental intervention, not as stand-alone solutions.

Chapter 13

Conclusion: synergy is the way forward

Contents

The original research questions and objectives of the research project (from Chapter 1) are re-stated here for easier referral. The main aim of this thesis is to describe, develop and evaluate an alternative model to the more traditional, reductionist framework for the management of overuse injuries. The main research question is:

> How can systems thinking and data-driven computational modelling enhance the management and prevention of overuse injuries?

with sub-questions:

1. What components constitute a systems thinking framework in sport?
2. How can the systems thinking perspectives be instantiated through computational applications?
3. How can the data from runner's wearables contribute to computational applications in sport?

13.1 Objectives

The specific objectives of the study were completed as follows:

1. *Contribute to a theoretical and practical foundation for a ST perspective in sport.*
   The ST rationale and paradigm were unpacked in Chapters 6 and 7, whilst uniquely applied to sport in general in Chapter 9.
2. *Introduce and develop the concept, the RCAS, as the combined biomechanical and physiological micro-systems of the runner and their physical, spatio-temporal environment.*
   The RCAS was described in a qualitative ice-berg model in § 10.1 and the rCLD in §

10.2. These descriptions built on the generic systems archetypes which were mapped to sport applications in § 9.1.2.

3. *Instantiate a ST framework in sport science with a practical application in the form of a computational modelling tool of the RCAS, using ABM and SD.*
   The dynamic, working hybrid simulation model was presented in § 10.3, followed by the outcomes from applied interventions to find leverage for long lasting change in § 12.2. The simulation model was evaluated in Chapter 11.

4. *Leverage large data sets generated by RWs (smart watches, activity trackers and running watches) to parameterise the synthetic population in the models.*
   Appropriate data from the RW were analysed in Chapter 8 using data mining techniques. The values of cadence, HR and the surface type were synthesised interactively as input parameters for the simulation model.

13.2 Implications for research and future work

The instantiated ST framework transcended reductionist thinking to arrive at causal, closed-loop thinking by presenting the RCAS as a pressurised plumbing system, in the form of a hybrid agent-based and SD simulation model. Understanding where the accumulation points in the system are and how the pressure may build up, is a practical analogue for the gradual onset of RROIs. Nonetheless, the simulation model is an abstraction of reality, and does require further work to improve. Streams of future work are arranged according to the selection of endogenous system variables and refinement of the simulation's mechanics.

13.2.1 Variable selection and inclusions

The simulation model was constructed on one athlete's data from their RW, and tested using two other athlete's data as input. A larger sample size is suggested to further refine the model's parameters and mechanics as well as expanding the levels of analysis to both lower levels of detail on a physiological level and higher, collective levels. The RW data collection process (downloading and extracting the container files from *GarminConnect*) is tedious and time consuming, limiting the amount of data that was extracted from participants. Expanding the sample size must consider the time cost to download and extract the data, or find an alternative that would accelerate data collection. Other open-source platforms are available to collect data from disparate athletes on a large scale (specifically, *Strava*), although the researcher would not have the same amount of control over data extraction as with the manual process through *GarminConnect*.

Currently, only the training load in terms of the impact absorbed is utilised in the model. The training load calculations (Equations 10.7, 10.8, Algorithm 1) took both primary and secondary data to derive input values for variables. Cadence and surface type, as extracted from the RWs, are utilised as primary data for the training load. Secondary data are the VO and leg stiffness from published work (Ferris and Farley, 1997). Empirical work is suggested to generate only primary data for input: to find the runner's VO and leg stiffness per surface type, alongside the existing cadence data from the RW. Other variables to consider for inclusion are psychological and socio-economic factors, environmental factors (specifically, relative humidity, slopes, and altitude), and resource constraints. Energy balances through nutritional intake may also be considered, which may form the bridge to socio-economic aspects. The data collection process for nutritional intake would have to be carefully designed with a sport nutritionist involved.

The classification framework to sample HR based on a cadence level (Algorithm 2, § 10.3.4) proved useful to link the biomechanical, cyclical workload to a CVS response. The cardiac workload is not quantified in this model, although the HR zone (based on the sampled HR value) is used in conjunction with the external temperature to gauge the delay between fibres in disarray and restored integrity (Equation 10.2). The formulation of this delay may be extended to leverage (if available) the HRV, or other recovery metrics that are obtainable from a RW. Including predicted values for the VO_2-max and the RE in the model may be considered to better gauge the CVS response or cardiac workload. However, more specialised data collection methods will be required.

13.2.2 Refinement of the simulation model

As per model characterisation in Sterman (2000), the simulation of the RCAS is a reflective model. The objective of the model is not to provide a final, definitive, analytical answer, but to facilitate learning the fine balance between equilibrium and disequilibrium in the body as a result of training, over the long term. The simplicity of the simulation to model the behaviour of the overall structure lowers the risk for mental overload, although it may not expose all assumptions necessary. As part of the learning experience, the model invites inquiry, multiple viewpoints, and more experimentation (perhaps re-drawing the boundary to include current exogenous variables). Empirical work is suggested to more accurately quantify the rates of the flows in the model, particularly the micro-damage rate and the entropy threshold. Other mechanical actions in the model to consider are:

- Refining the limits of the fitness and/or damage zones.
- Extending the time in the damage zones.
- Raising or lowering the limits of the entropy threshold.
- More structure specific modelling of tissues (bone and soft tissue fibres).

The current structure of the RCAS itself, the structure of the stock-and-flow SD component, and the decision rules by which the agent-based component behaves are also subject to alternations. The extension of the model to other system levels is encouraged. A higher level would be that of a stratified population of runners, organisational, international, and even industry level. Lower levels would require more in-depth physiological parameterisation. Both extensions would require more runners (a larger sample size) and a re-think on data collection.

13.2.3 Validation through expert opinion

An external validation of the simulation model as an instantiation of the ST principles of the RCAS will be valuable to the research community. A future study is proposed for this purpose. Such a study would involve a qualitative, interview-based approach with practitioners, sport scientists, and potentially also athletes.

13.3 Clinical relevance: the practical applications

Externalisation of mental models is a challenging prospect. This project attempted to externalise a mental model to balance training and physiological fitness, first for sport in general and then as a focussed, conceptual simulation exercise applied to running. The clinical relevance of this research project is not the efficacy of the interventions applied in the simulation model. It is about the methodological thought process to arrive at the intervention, and extending the thinking to hypothesise how the intervention affects future behaviour. The value of the ST framework for practitioners lies in the methodological thinking to discern between fundamental and symptomatic interventions in the dynamic, complex solution space. The solution space consists of multiple, interacting levels (complex) and has inherent time components (dynamic). For instance, in this thesis, the argument was made earlier that new running shoes may be a symptomatic solution to poor running performance and that biomechanical interventions constitute a fundamental approach (§ 9.2). Yet, an alternative view may be that, for some runners, the *correct* shoe forms part of the fundamental solution.

The ST framework promotes the process to find optimal leverage through changing the elements of structure (through their interactions, how they are connected or altering individual properties) instead of finding solutions to address symptoms of problematic structure. Finding optimal leverage demands causal, closed-loop, dynamic thinking. The system archetypes is a good starting point to articulate sports problems into causal, closed-loop themes. These themes may be customised into individual, dynamic hypotheses, as was done for the shoes in the growth-and-underinvestment system (§ 9.2).

Data from RW can be put to work in mathematical expressions to quantify training loads that can be interpreted in a composite value with practical meaning, instead of separate data streams. The biomechanical data from the RW were connected in a mathematical expression (the training load) to quantify (to some extent) the behaviour of the connected elements in the RCAS (see Equations 5.27 and 5.28 for formulation and § 10.3.4 *Selecting training parameters and the main input function: pulsed training load* for application). It was shown it is possible to utilise the data in a novel way: in aggregated measures and as part of a computational application, yet each variable maintained its significance.

The practical simulation model showed empirically how fundamental solutions and respecting the delays inherent to the RCAS resulted in sturdy structure formation, delivering an athlete who is both fit and healthy. The parameterisation of the simulation by data from RWs adds to the representativeness and preciseness of the model. The RW data allow subject-specific modelling of the runner at the individual level, with some information of their immediate environment. The practical illustration of the ST framework focussed on running, but any sport or athlete may be presented in a similar manner, although parameterisation mechanisms would differ.

13.4 The value of the design science research methodology

The DSRM guided the overall process of creating the ST framework and instantiating the ST perspectives through the simulation of the RCAS in a hybrid model. The rigour cycle of DSRM warranted the researcher to first evaluate the existing knowledge bases for both theories regarding running-related phenomena and solutions to RROIs. When current solutions were found inadequate or sparse, the researcher further engaged with existing methods to design the ST framework. Furthermore, the rigour cycle ensured that the theories and methods used to construct the RCAS are substantiated and logical. The relevance cycle aided in establishing and understanding the need for ST in sport, more specifically, the shift from linear, cause-effect thinking to closed-loop causal thinking and incorporation of system levels. The design cycle consisted of building and evaluating the qualitative, descriptive ST models (the ice-berg model, the sporting system archetypes, the rCLD), the data mining process (to quantify appropriate input parameters), and the simulation model itself. Evaluation consisted of an iterative process between the phases, as well as a formal process for the simulation model with the guided model tests as explained in Sterman (2000). The evaluation provided feedback to the researcher to modulate or improve the current designs.

The knowledge base has been augmented with a framework and an instantiation of the ST principles to support reductionist studies in the sport domain. This framework may be followed by other researchers and practitioners to develop more frameworks or enhance the current version. As time progress, additional frameworks and practical applications may collectively culminate in meta-artefacts such as design processes aimed at performance enhancement or injury prevention, supported by ST principles. The change and impact cycle is yet to realise, however the two publications (§ 1.7 *Publications*) prior to completion of this dissertation aid in the dissemination of the ST framework in sport.

13.5 Personal reflection from the researcher

The researcher found the project to be both challenging and rewarding. Challenges encountered are related to the methodological process of the study as well as technical difficulties regarding software compatibility and use of applications to build the models. The transdisciplinary nature of the study demanded from the researcher to be well-versed with the sport science domain, as well as the engineering techniques applied. The bachelor degrees (Physiotherapy and Engineering) the researcher obtained earlier provided a strong foundation from where the knowledge bases could be investigated, combined with faster absorption and interpretation of domain literature. Yet, the mental effort to engage the two domains and extract the relevant knowledge in a meaningful way was exceptionally challenging. This study did not only require an application knowledge of sport science and running, but demanded understanding on a deeper, structural level at which the engineering applications are aimed.

The training load function is considered by the researcher as the fundamental mechanism between the tracking data, the runner as a spring-mass system, and the simulation model. This function abstracts the attributes of a runner into a mathematical expression, which culminate in the main input function for the simulation model. Although simple in its form and well-known in physics, reaching this function and its purposeful application in the simulation model, was the most elusive aspect of the entire project. Simultaneously, operationalising this function in the simulation model to drive the continuous input was also the most rewarding moment of the research journey.

The researcher valued the feedback received during the academic writing process. Feedback from supervisors, reviewers (of the two publications), and attendants at the SD conference in Stellenbosch all contributed meaningfully to the build-and-evaluation cycle of the final dissertation. Substantiation of facts and methods (even sometimes assumed to be well-understood to the point of 'common knowledge') in literature remains crucial to the integrity of any research project. Nonetheless, even more so in a transdisciplinary project such as the one presented in this dissertation, where readers from different fields are introduced to unfamiliar concepts or theories.

There are potential direct outflows from the DSRM, in that another researcher may take upon themselves aspects of the proposed future work; alternatively be inspired by the ST framework and embark on a similar project. Practitioners may apply the principles founded in this thesis to their own clinical practice toolbox towards management of athletes, patients or their own participation in sport. Improved clinical outcomes from applying ST principles remain subject to implementation and active attention paid to delays. Indirect consequences are expected to only present over time. As with any new technique, ST principles must be practised consistently for the mind to learn to search abductively and 'close the loop'. Adopters and users of ST must remain cognisant not to overcomplicate problem structuring through addition of variables, or whilst mapping the CLD, which will not suffice to find leverage but may only exhaust the mind's efforts. This may result in resistance to and possible abandonment of the

ST methods in sport. Should ST find useful, practical application in sport science, it may in the future be incorporated in undergraduate training programs so that it becomes part of the basic toolbox of any sports-related practitioner.

13.6 Conclusion

The ST framework illustrated that both analytical, reductionist thinking and synthetical thinking at the system level is required to understand complex sport injuries better. A ST framework constitutes of qualitative, descriptive models, data analysis and quantitative computational modelling of the system. The domain of sport science was taken through the qualitative process of system deconstruction using ST tools, followed by the synthesis and simulation of the RCAS in a closed-loop thinking mechanism. The qualitative ST models first decomposed the RCAS into the interacting constituent elements in parallel, and then practically facilitated the causal thinking about training, structure formation and damage accumulation over time in a simulation model.

Athletes train and condition their body for optimal performance on all levels of participation, be it to advance athletic frontiers or simply to remain fit and healthy. The scientific disciplines of ST and computational modelling have attributable roles to play in sport science, specifically towards training and conditioning to maintain athletic health and avoid injuries. This thesis has shown that there is some 'method in the madness' in the synergy of reductionist and abductive thinking: the big-picture (the forest) matters as much as the detail (the individual trees), although it is challenging to keep both in mind simultaneously.

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Appendix A

Digital footprints of family members

A.1 Athlete 2

Athlete 2 is a highly capable marathon runner.

A.1.1 General

Table A.1: General statistical measures of dispersion for running data, athlete 2

Metric	Mean ± st.dev	Range (min, max)	IQR (25^{th}–75^{th})
Heart rate (*bpm*)	145.43 ± 14.72	111, 176	135 – 157
Cadence (strides per min)	83.50 ± 4.13	74, 100	81 – 84
Pace (min/km)	4.80 ± 0.94	0.67, 8.32	4.43 – 5.29
Distance (km)	12.67 ± 14.37	1.85, 90.68	6.27 – 11.13

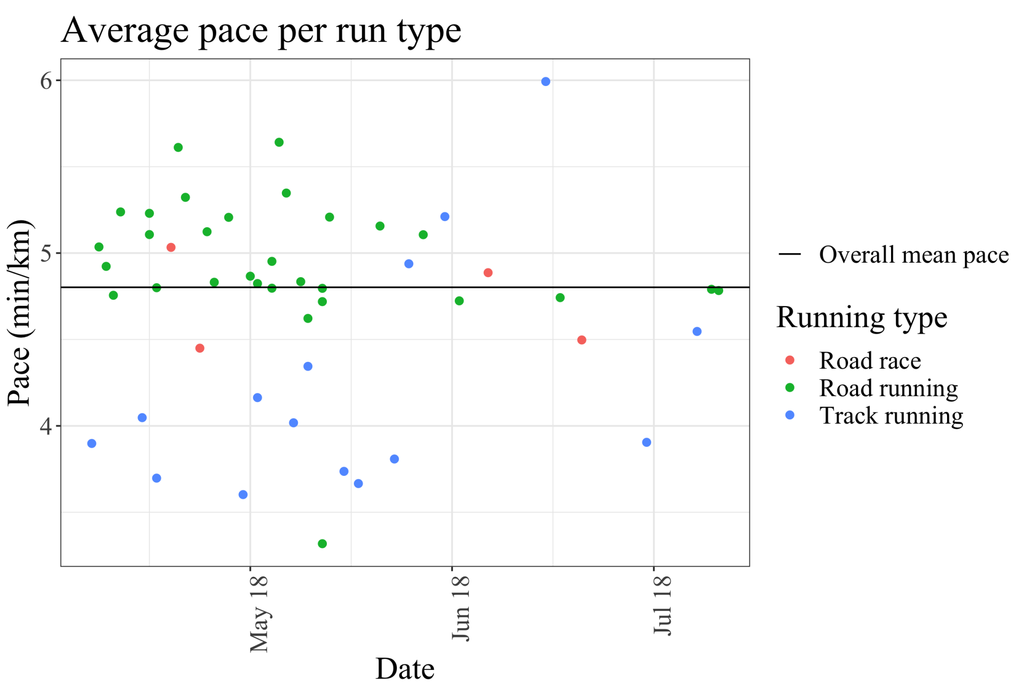

Figure A.1: Pace performance, athlete 2

A.1.2 Outliers

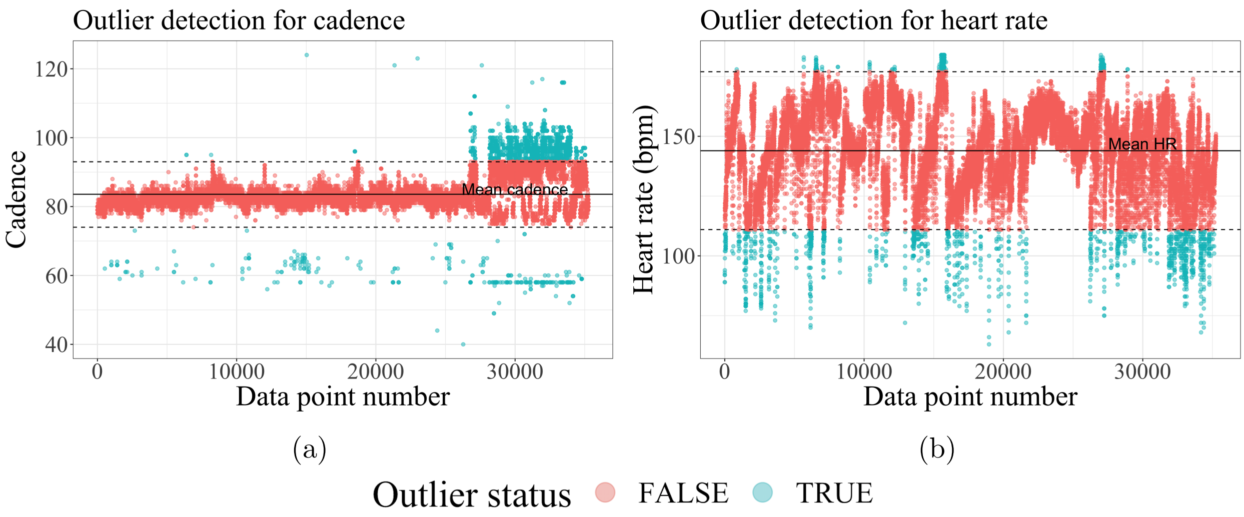

Figure A.2: Distribution based outlier detection for cadence and HR

A.1.3 Distribution of parameters

Table A.2: Input probability mass function for run types, athlete 2

Run (surface) type	**Probability**
Road race (rc)	0.0263
Road running (rr)	0.579
Track training (tt)	0.395

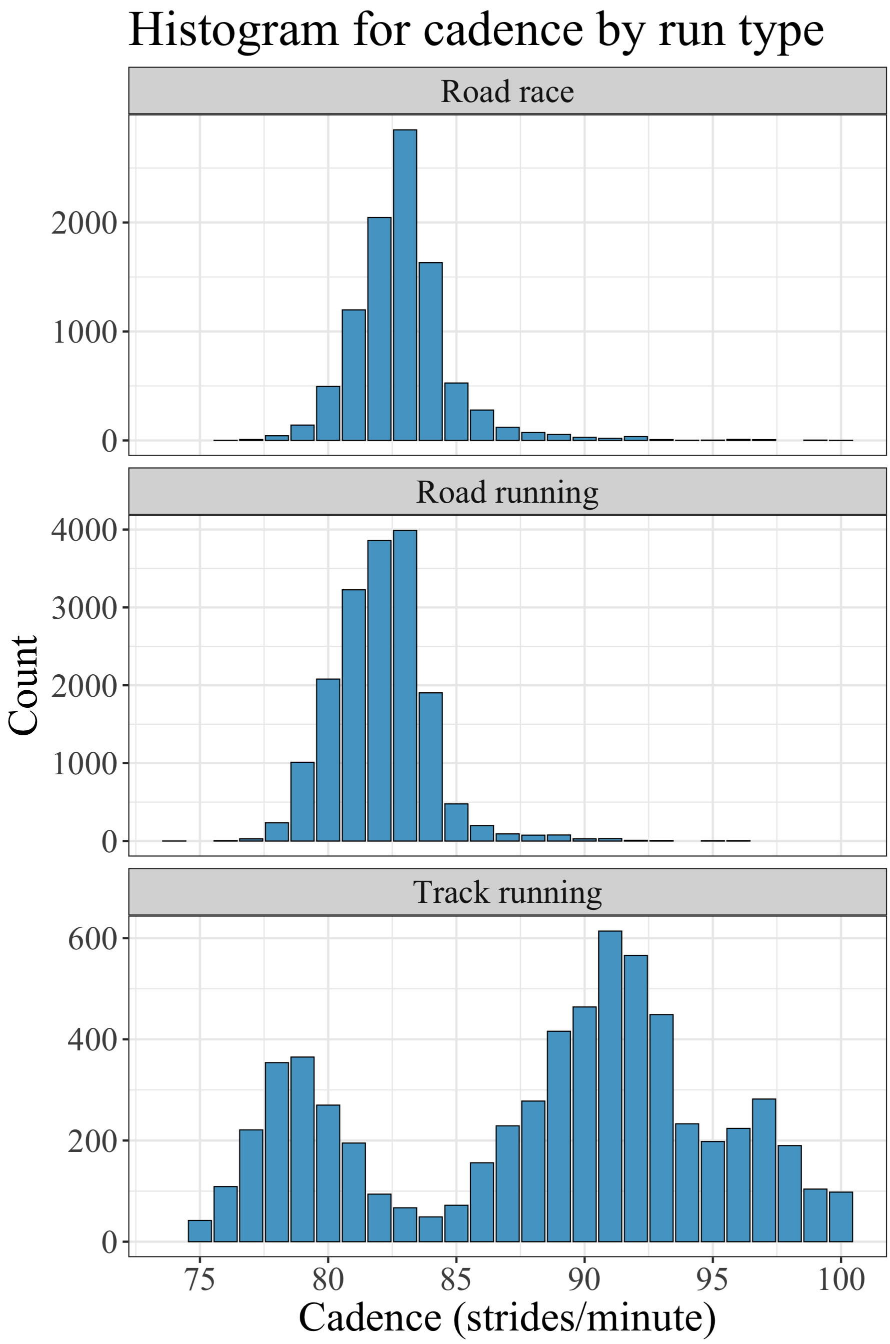

Figure A.3: Histogram for cadence, athlete 2

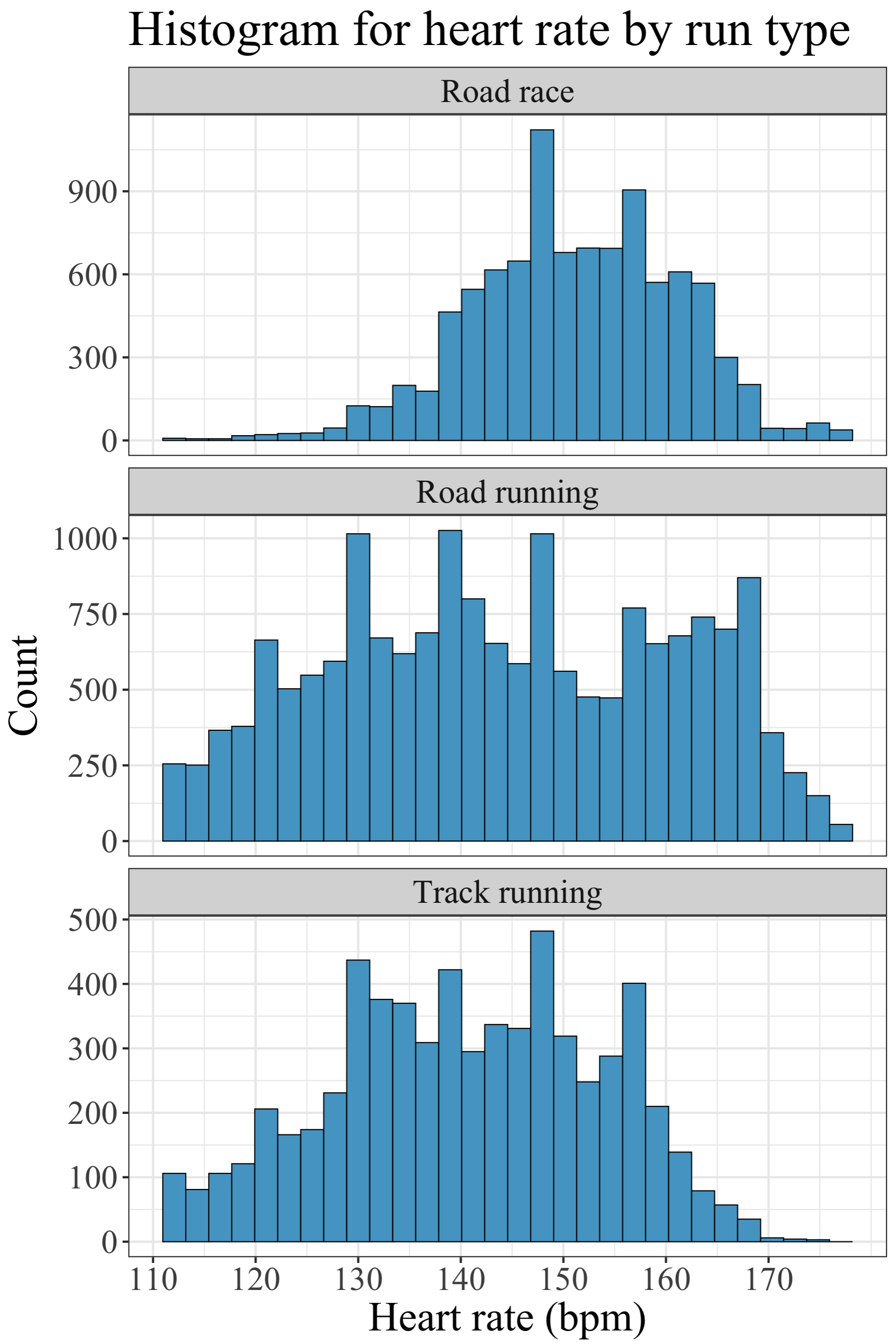

Figure A.4: Histogram for heart rate, athlete 2

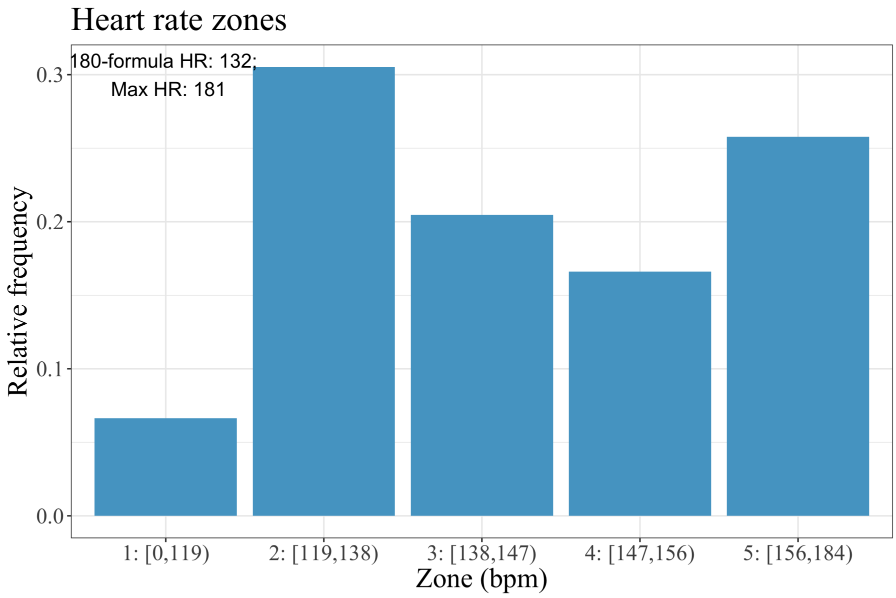

Figure A.5: Heart rate zones and limits, athlete 2

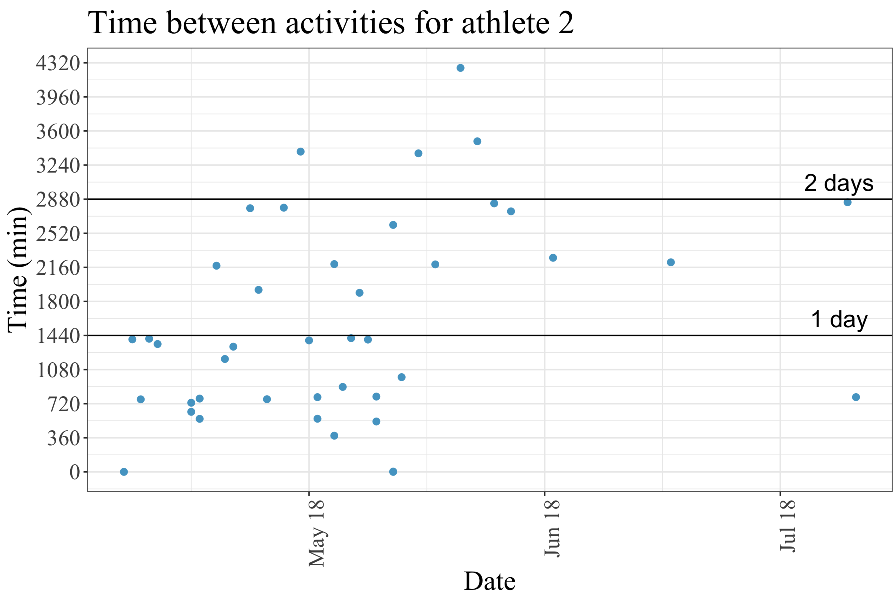

Figure A.6: Time between activities, athlete 2

Table A.3: Input values for parameters per surface type, athlete 2

Run (surface) type j	**Cadence: mean ± st.dev** (steps.s^{-1})	**VO: mean ± st.dev** ($meter$)	**Leg stiffness** k (kN/m)
Road race (rc)	2.779 ± 0.0263	0.12 ± 0.012	20
Road running (rr)	2.732 ± 0.579	0.12 ± 0.012	20
Track training (tt)	low: 2.644 ± 0.0757	0.06 ± 0.006	40
	high: 3.07 ± 0.1154	0.06 ± 0.006	40

A.1.4 Heart rate and cadence interaction framework

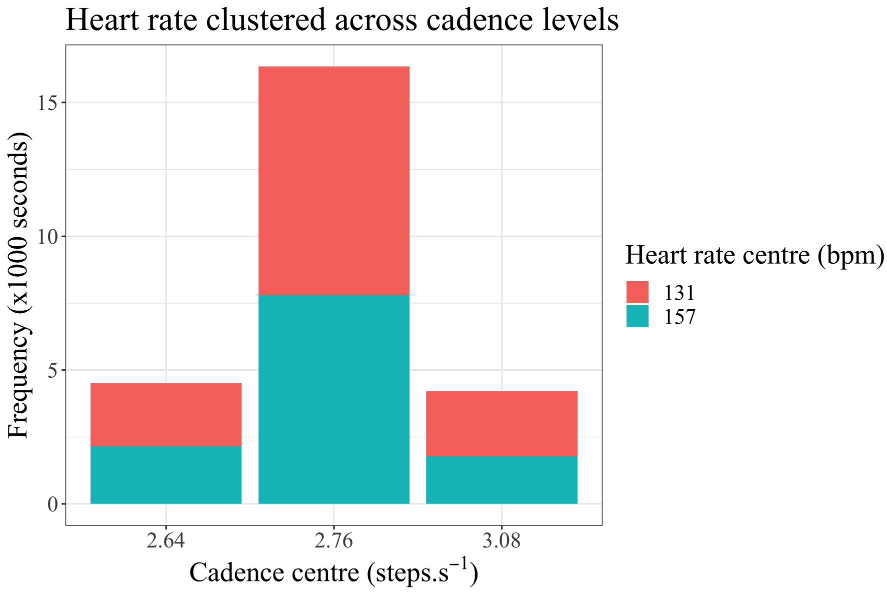

Figure A.7: Proportional distribution of heart rate clusters per cadence level, athlete 2

TableA.4 provides the probability distribution of the cadence and HR clustering classification framework.

Table A.4: Probability mass function for heart rate and cadence levels, athlete 2

Cadence level	Heart rate level	Probability
Low	Low	0.5203
	High	0.4797
Mid	Low	0.5226
	High	0.4774
High	Low	0.5799
	High	0.4201

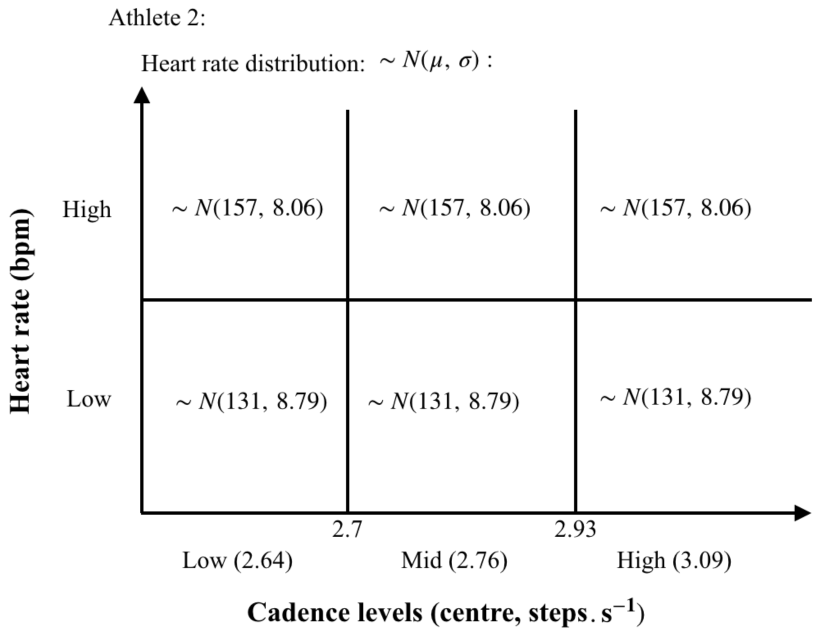

Figure A.8: Parameters for heart rate from cadence classification, athlete 2

A.2 Athlete 4

Athlete 4 is a professional trail athlete, competing in ultra-distance races.

A.2.1 General

Table A.5: General statistical measures of dispersion for running data - athlete 4

Metric	**Mean ± st.dev**	**Range (min, max)**	**IQR (25^{th}–75^{th})**
Heart rate (*bpm*)	148.64 ± 9.98	122, 172	142 – 156
Cadence (strides per min)	87.45 ± 3.39	77, 97	85 –89
Pace (*min/km*)	5.35 ± 1.09	0.82, 8.33	4.72 – 6.00
Distance (*km*)	12.86 ± 11.50	0.12, 297.29	6.04 – 14.70

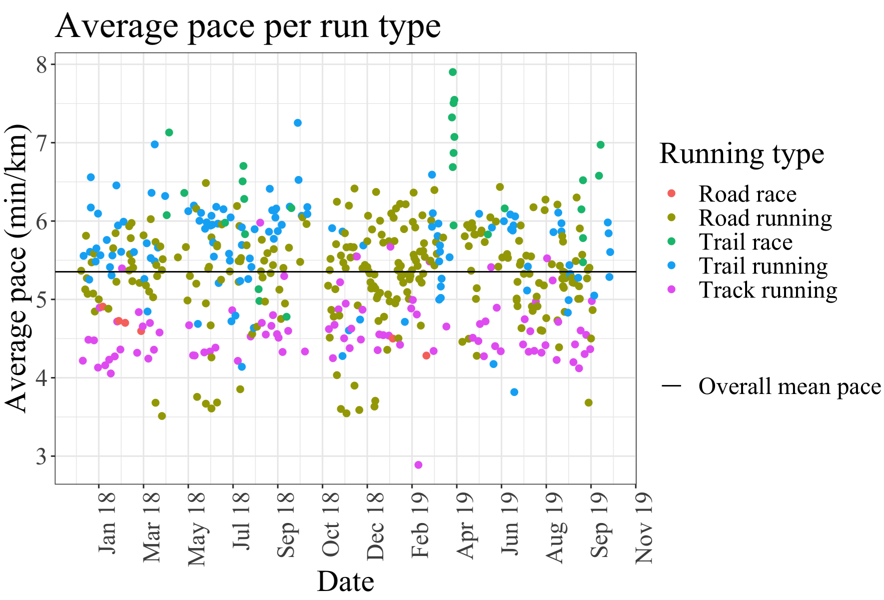

Figure A.9: Pace performance, athlete 4

A.2.2 Outliers

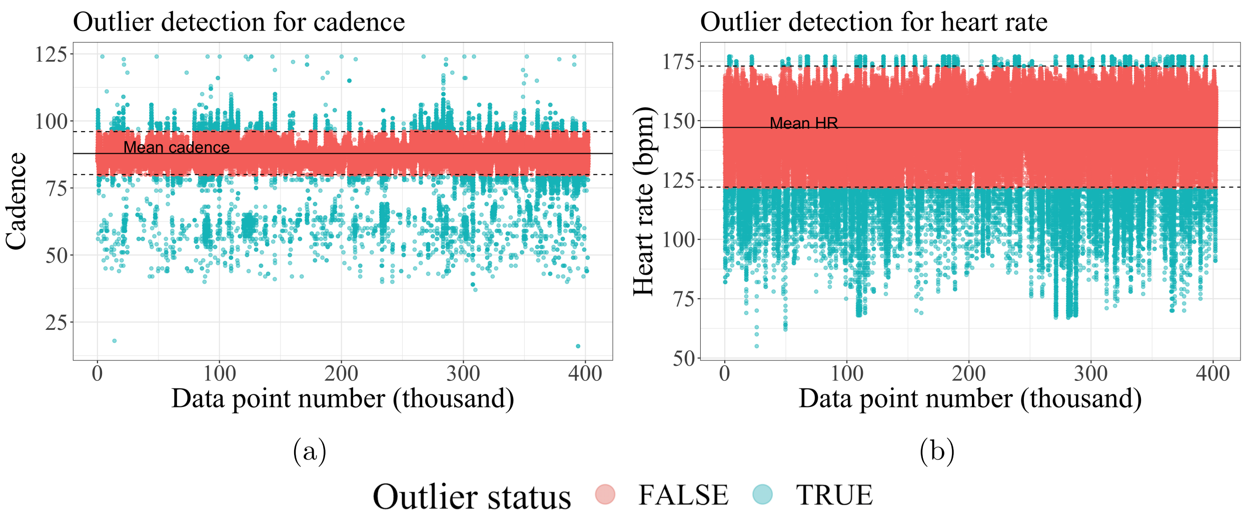

Figure A.10: Distribution based outlier detection for cadence and HR, athlete 4

A.2.3 Distribution of parameters

Table A.6: Input probability mass function for run types, athlete 4

Run (surface) type	Probability
Road race (rc)	0.0032
Road running (rr)	0.535
Trail race (tc)	0.003
Trail run (tr)	0.161
Track training (tt)	0.297

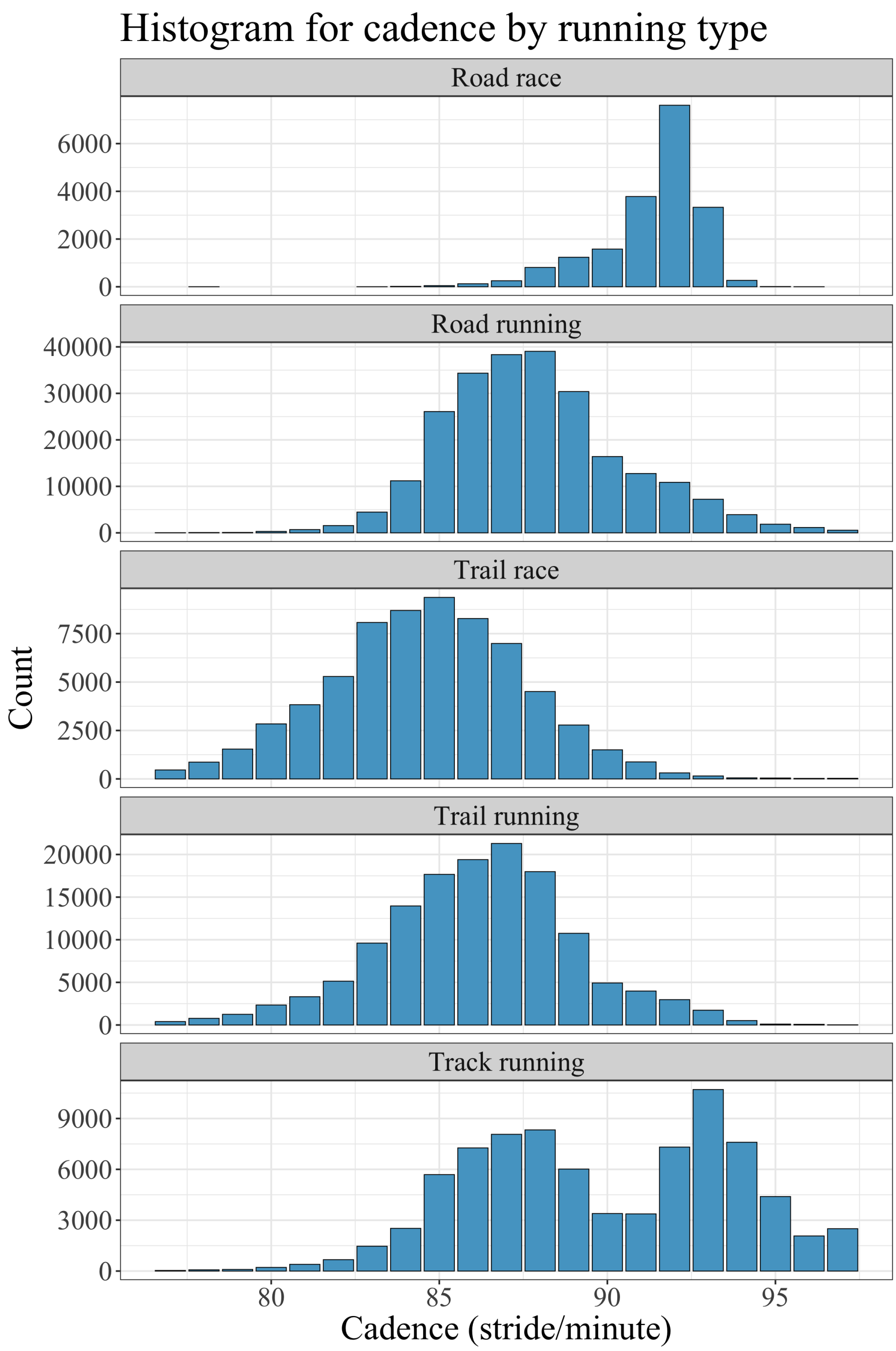

Figure A.11: Histogram for cadence, athlete 4

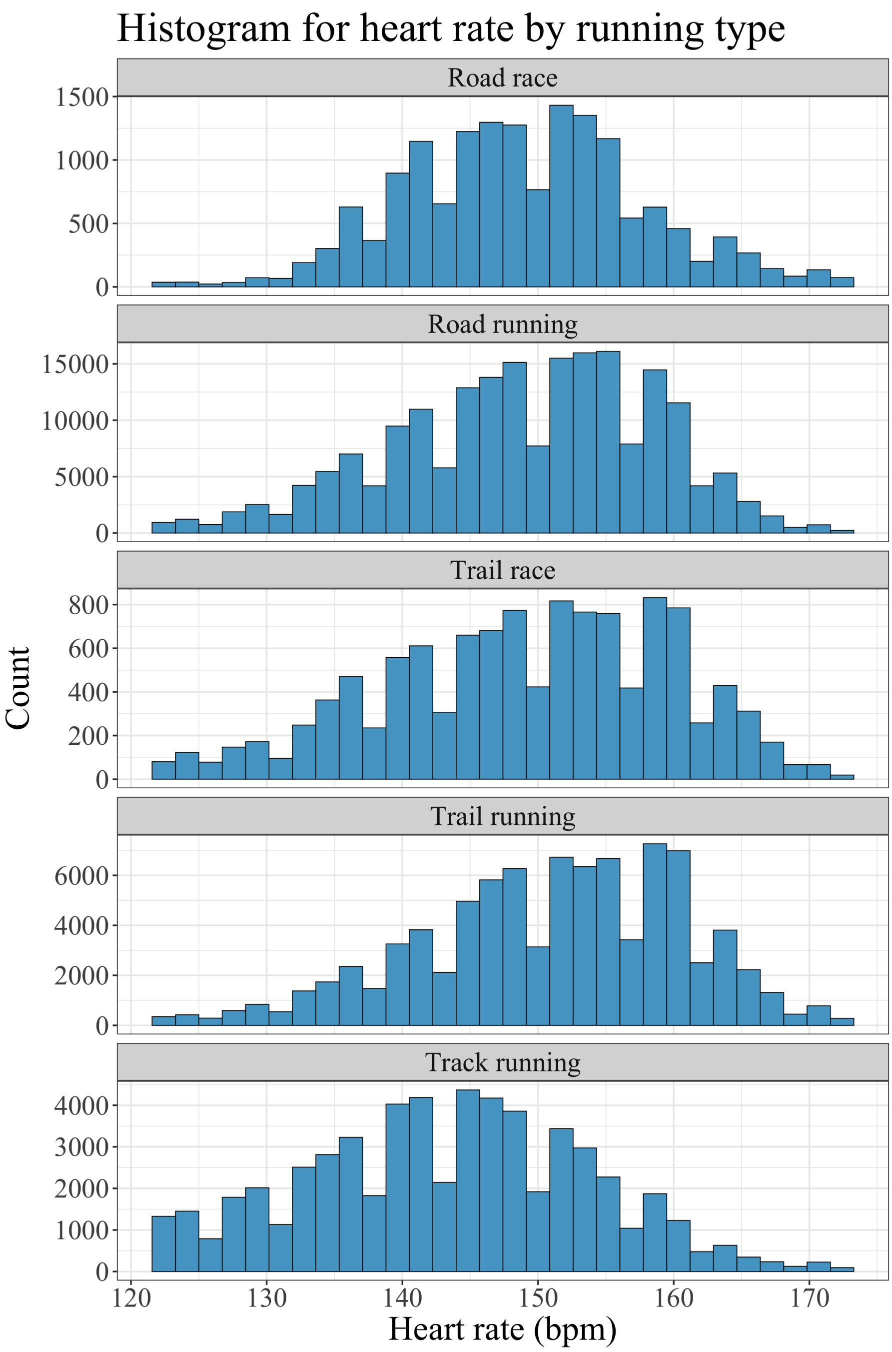

Figure A.12: Histogram for heart rate, athlete 4

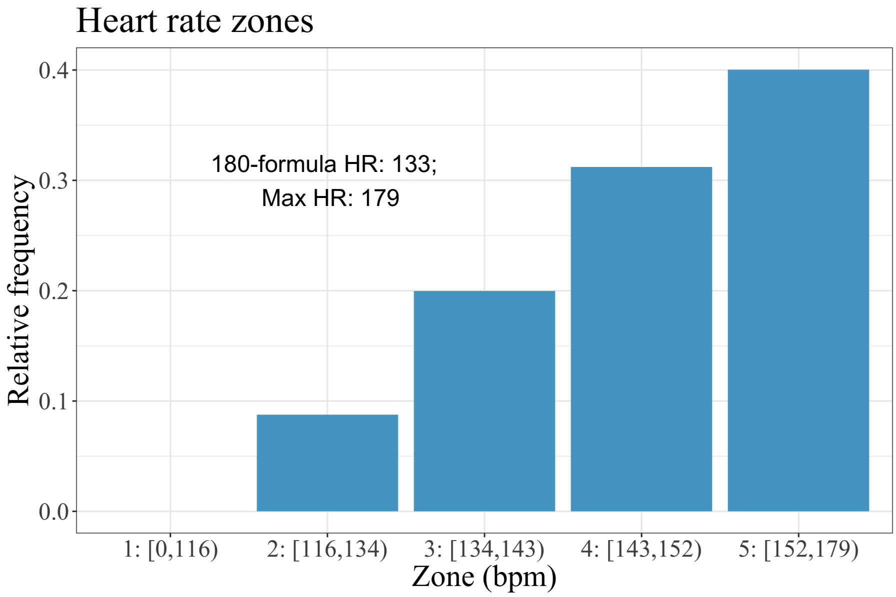

Figure A.13: Heart rate zones and limits, athlete 4

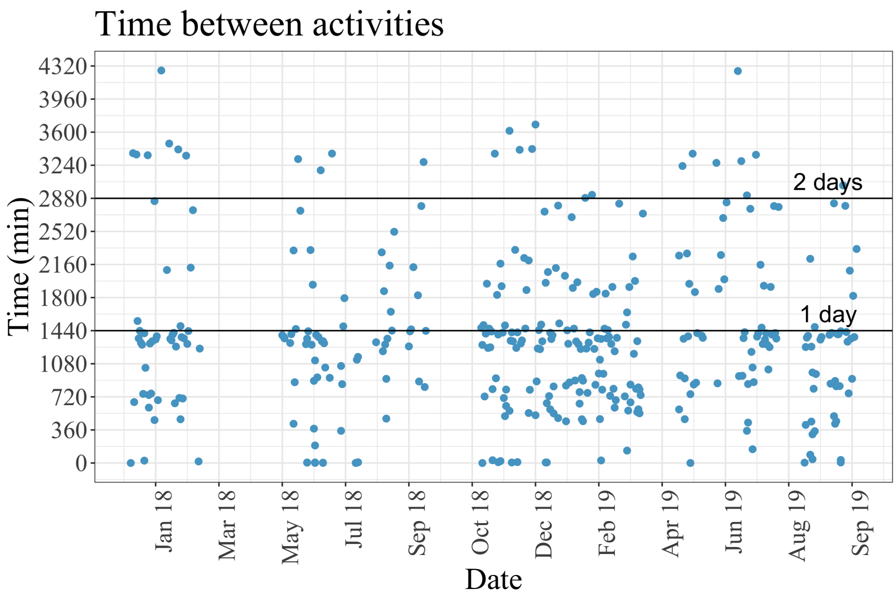

Figure A.14: Time between activities, athlete 4

Table A.7: Input values for parameters per surface type, athlete 4

Run (surface) type j	**Cadence: mean ± st.dev** (steps.s^{-1})	**VO: mean ± st.dev** ($meter$)	**Leg stiffness** k (kN/m)
Road race (rc)	3.040 ± 0.053	0.12 ± 0.012	20
Road running (rr)	2.928 ± 0.09	0.12 ± 0.012	20
Trail race (tc)	2.993 ± 0.047	0.08 ± 0.008	30
Trail run (tr)	2.873 ± 0.1	0.08 ± 0.008	30
Track training (tt)	low: 2.883 ± 0.0601	0.06 ± 0.006	40
	high: 3.102 ± 0.0614	0.06 ± 0.006	40

A.2.4 Heart rate and cadence interaction framework

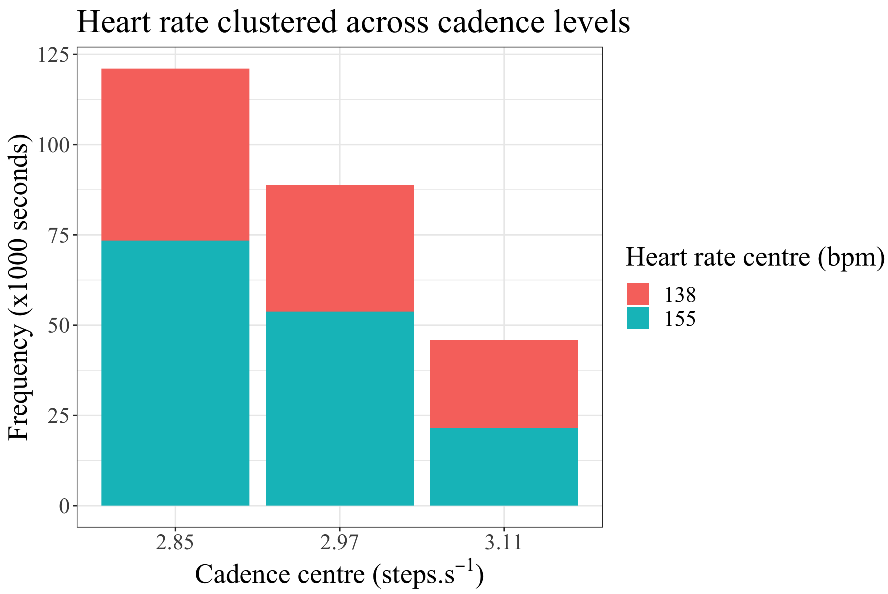

Figure A.15: Proportional distribution of heart rate clusters per cadence level, athlete 4

TableA.8 provides the probability distribution of the cadence and HR clustering classification framework.

Table A.8: Probability mass function for heart rate and cadence levels, athlete 4

Cadence level	**Heart rate level**	**Probability**
Low	Low	0.393
	High	0.607
Mid	Low	0.394
	High	0.606
High	Low	0.529
	High	0.471

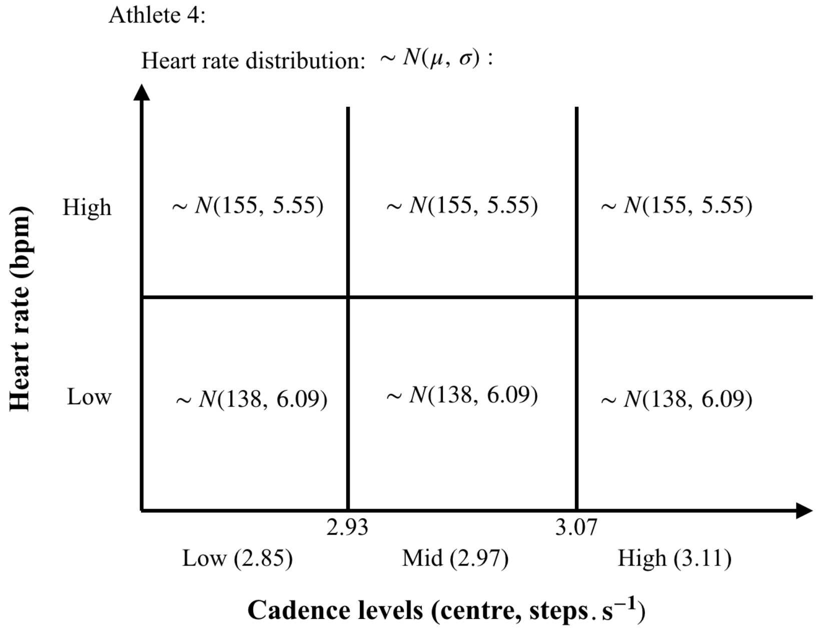

Figure A.16: Parameters for heart rate from cadence classification, athlete 4

Appendix B

Ethical governance: data management and study virtues

The study received ethical clearance from the Stellenbosch University's Research Ethics Committee: Social, Behavioural and Education Research (SBER), with project number 10124.

B.1 Risk-benefit equation

Minimal risk: there is no anticipated discomfort or stress besides what the athlete experiences in their day-to-day lives. There is no interruption of their daily activities. The athlete is under no obligation to train, race or to record future activity if they do not want to. The researcher acquire the data that is available. The runner was advised to refrain from physical activity if they feel unwell or are injured, and to consult a medical professional.

Benefit: indirect, as the eventual utility of the application may add value to the athlete's self-monitoring of their conditioning over time. A coach and the sports medical practitioners may gain insight into the dynamic behaviour of sport performance and injuries, in order to better manage the athlete for career longevity.

B.2 Data collection from runners wearables

The same athletes who partook in the research by Vermeulen (2018) were approached via telephonic communication. These athletes are members of the same running club. Although this may introduce bias into the data, the study is concerned with the usability of the data and the method on how to use the data. The bias was therefore not a threat to the outcome of the study. The time line of the data collection is January 2017 (or the earliest available data from the athlete) to September 2019.

Athletes continued to run, train and race with no intervention from the researcher. Written, informed consent was obtained from the athletes before data were collected.

B.2.1 Collection procedure

The data collection procedure is simple:

1. After a run, runners sync their data from the device to the fitness application (Garmin Connect) as they normally do.

2. At 1 month intervals, the researcher access their on-line profile (with written permission from the athlete) and download the GPS container files with the required data from the

running sessions of the past month. This file is accessed via the *Option* icon on the specific activity session and selected as *Download tcx format*. The researcher downloaded the retrospective, historical activity data during the first access session.

3. Each run session is visually inspected on Google Earth to classify the running activity based on the location. The on-line application, *GarminConnect*, has this functionality built-in so it is easy to inspect the route and classify it as road, track or trail. This is a critical step in data validation, as it forms part of the environmental factors that impact running.

4. Once downloaded, container files are stored on a laptop.

5. The GPS container files are named per the following convention: *athlete ID - run type - date of activity - activity number*. The activity number is auto-generated from the on-line application. The date of the activity is captured in the format yyyy-mm-dd. The athlete ID is the unique identification number given to each athlete to ensure anonymity of the data. The type of running activity can be:

    - rr for road running;
    - tt for track training;
    - rc for a road race;
    - tr for trail running;
    - tc for trail race.

6. The data in the container files is extracted and written to a database using a combination of the programming languages *R* and *SQL*.

For example, the file

7rr2018041124578398

is the GPS container file for athlete number 7 on 11 April 2018 for a road run session. The last eight digits constitute the file name as the activity number originally generated by the application. This naming convention will be used in order to manage the data per athlete, run type and dates. This constructed file name will also imported together with the data set for the sake of trace-ability and data management control.

Because the runners represent archetypes in the simulation model, the sample size was kept small. However, the data set per individual athlete was large, expected to cover more than two years of running data. Data that is to be collected from the athlete:

- The GPS container files include the GPS location (latitude and longitude), the altitude, a date-time stamp, distances covered, speed, cadence, and heart rate for nearly each second over the duration of the run activity.

- Other data directly extractable from the application: age, sex, height, VO_2 - max (if provided by the device). The athlete may also choose to provide this data on the consent form.

B.2.2 Informed consent

In order to obtain informed consent, the following steps are taken:

1. The athlete is provided with the consent form that also contains the information leaflet.
2. The study is explained to the athlete and they have time to ask questions.
3. The athlete may take the consent form and study the information therein during their own time, before making a decision.
4. When they decide on their participation, they inform the researcher. On the form they may indicate either "allow" or "deny" the researcher to use their running data.
5. Both the athlete and the researcher sign the form, and the athlete receives a signed copy of the form.

If the athlete granted consent, they provide the researcher with access to their on-line profile. The details to their profile are kept safe and confidential, and access frequency is kept to a minimum.

B.3 Ethical considerations

The following considerations are in place to ensure a sound ethical study and protection of the participants:

1. Participant characteristics
   (a) Healthy (in all definitions of the word, including mental), adult (older than 18 years) athletes who partake in middle to long distance running.
   (b) Requiring no professional supervision.
   (c) Currently using a runner's wearable and is familiar with how to use it.
2. Voluntary nature
   (a) The athlete was informed that their participation is completely voluntary and that they should not partake if they do not want to. There are no negative consequences for the athlete if the decline to participate.
   (b) The athlete was given all the necessary information and review time to provide informed consent for their data to be used in the study.
   (c) The athlete kept a copy of the signed informed consent (or dissent) form.
3. Interests
   (a) The study forms part of the researcher's fulfilment duties for PhD Eng: Industrial and is funded by the researcher herself as well as a scholarship from the Council of Scientific and Industrial Research.
   (b) There is no incentive for participants.
   (c) There is no sponsor with technological or financial interest involved.
   (d) There is no personal conflict of interest for the researcher or other parties that promote training methods or race tactics, because the athletes will carry-on with their own training program and race tactics as they see fit.

(e) The athlete is also under no obligation to train or participate in races.

4. Data characteristics

   (a) The raw data do not include any personal information that may identify the athlete.
   (b) The researcher did not perform any measuring of the data herself using instruments or apparatuses, but only used the data as generated by the wearable device.
   (c) The manufacturer of the wearable devices have declared that the devices are not medical devices and that the data is not intended to be used to diagnose or cure any disease. The data is to be used to encourage a healthy lifestyle. This research study is also not intended to diagnose or cure injuries.
   (d) Participants' anonymity was ensured and all their data be safeguarded.
   (e) Only data from participants who have granted informed consent were used in this study.
   (f) Processed data with codified athletes is separated from the athletes' identities by location, that is the processed data is stored on one device in location A and their names are kept safe on another device in location B.
   (g) Both raw and processed data are backed up to the cloud as well as external hard drive devices (still separated from any identities). Access to these locations is password protected, with the passwords being strong and updated.
   (h) Consent forms were signed electronically and are kept safely on storage devices.

5. Transparency and publications

   (a) Participants have access to all research and information relating to them personally at any stage during or after completion of the study, including the findings and conclusion of the study.
   (b) All data and results will be and remain the property of the Stellenbosch University.
   (c) Result may be released as part of the dissertation for attainment of the Doctoral Degree in Industrial Engineering and published in selected scientific journals.
   (d) Participants were informed that they may withdraw from the study at any point of time, after which their data will be excluded from the analyses and the computational model.

From these considerations the researcher did not foresee any physical or psychological harm to the participants.

B.4 Container data file example

Table B.1: Structure of the table for raw data extraction with generated example data

	date_time	latitude	longitude	altitude_meters	distance_meters	heart_rate_bpm	speed_ms	cadence
10	2018-02-08T15:39:24.000Z	-25.848	28.140889	1442.8000	29.9899	155	3.46900	71
11	2018-02-08T15:39:25.000Z	-25.848	28.1408	1442.599	33.569	155	3.4930	91
12	2018-02-08T15:39:26.000Z	-25.84886	28.140816	1442.5999	37.4900	160	3.5799	91
13	2018-02-08T15:39:27.000Z	-25.84887	28.140780	1442.5999	41.3199	161	3.6319	91
14	2018-02-08T15:39:28.000Z	-25.8488	28.14074	1442.4000	45.0600	160	3.6530	90
15	2018-02-08T15:39:29.000Z	-25.8488	28.14070	1442.4000	48.7400	158	3.6579	89

Appendix C

Consent form

This consent form is similar to what was used in Vermeulen (2018). It has been adapted to suit the current research project.

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jou kennisvennoot • your knowledge partner

STELLENBOSCH UNIVERSITY
WRITTEN CONSENT TO PARTICIPATE IN RESEARCH

Title of research project	Systems thinking and data-driven computational modelling in sport: a (new) framework for the management of complex sport phenomena.
Reference number	ING-2018-10124
Principal investigator	Euodia Vermeulen
Address	Department of Industrial Engineering, Stellenbosch University, c/o Banghoek and Joubert streets, Stellenbosch
Contact number	083 420 8770
Email address	euodiav@gmail.com

Dear athlete,

You are invited to volunteer for a research study. I am an PhD student at the Department of Industrial Engineering at Stellenbosch University, and I require some (much needed) assistance from the running community with an important building block in my research study. This information leaflet is to help you to decide if you would like to participate. Before you agree to take part in this study you should fully understand what is involved. If you have any questions, which are not fully explained in this leaflet, do not hesitate to ask Euodia. You should not agree to take part unless you are completely happy about all the procedures involved. In the best interests of your health and fitness, it is strongly recommended that you discuss with or inform your personal doctor, physiotherapist, coach and/or other physical trainers of your possible participation in this study, wherever possible.

C.1 Purpose

The running research community have come to realise that we need a new approach to do research on, and with, runners like you. We want to make use of a different thinking methodology than the traditional research approaches have done thus far. To do so, we are going to combine a technique called 'systems thinking' and the data from your tracking device to better understand how the athlete's body works as a system in its unique environment. This study will open doors to better manage your sporting performance and avoid or manage injuries. We want to give athletes, coaches and other role players involved the opportunity to change the way they think about their running and their environment, so that they can train better and keep injuries at bay as much as possible.

C.2 Procedure

The procedure is very basic. You record your runs (training as well as races) onto your fitness tracker or watch and synchronise the data to your online profile and application on your phone as you normally do. This is all that is required from your side. The researcher extracts the data from your online fitness profile and stores it on a database. From there the data is read into a software program for the purpose of analyses and further work. I also need to ask you for the following information: age , your sex and the VO_2-max as provided by your device (if it is available). This information you may fill in at the end of the form, if you decide to participate.

C.3 Time

The researcher needs access to both historical recorded activities (dating back to January 2017, or the time you started recording activities onto the fitness platform, up to the completion of the data analyses phase of the study, which is expected at August 2020). This large range of data will provide a better understanding of the changes in the running performance over the included seasons, which is an important factor under consideration for the study.

C.4 Risks

You bear no physical or mental risk during your participation in this project, as there will be no intervention on training methods or race tactics from the researcher's side. You are under no obligation to run, train or race. When you are unable to run (due to injury, illness or whatever other reason) you are advised not to participate in running activities. You continue with your normal training program, race schedule and race tactics as you and/or your coach sees fit. The researcher merely requires the data from those training sessions and races from your online fitness profile. Your data will not be shared with any of the other participants. Your online profile details will be kept safe and the frequency of access will be kept to a minimum

C.5 Benefits and incentives

Participation in the research study is done on a voluntary basis. Although the researcher is forever grateful to you if you choose to participate, you will not receive compensation of any kind for you participation. However, you will be part of an exciting journey in the research field to advance understanding of athletes' dynamic performance and how we can better prevent and manage injuries.

C.6 Participation and withdrawal

You are free to withdraw from the study at any time, without any negative consequences. Should you withdraw, all your captured data and information will also be withdrawn from usage in the research and be permanently deleted.

C.7 Confidentiality

You, as an athlete will be anonymised using a unique code identifier. All records obtained in this study will be regarded as strictly confidential. Neither the raw data nor the final results will contain any information which may identify you. You have access to all your data as well as the results pertaining to you.

It is required by the University that the results be published in a final report in the form of a thesis as well as an article in a journal. The results will be published or presented in such a fashion that you remain unidentifiable. The raw data and final results will be kept safe in a local database, on a laptop that is password protected and secured. The laptop's security features are updated regularly. The data will be backed in a secured cloud environment and on separated hardware discs.

C.8 Contact information

If you have any questions concerning this study you should contact: Euodia Vermeulen cell: 083 420 8770 or email euodiav@gmail.com. My supervisor (Prof. Sara Grobbelaar) may be contacted at ssgrobbelaar@sun.ac.za

C.9 Rights of research participants

You may withdraw your consent at any time and discontinue participation without penalty. You are not waiving any legal claims, rights or remedies because of your participation in this research study. If you have questions regarding your rights as a research subject, contact Ms. Maléne Fouché (mfouche@sun.ac.za / 021 808 4622) at the Division for Research Development. You have the right to receive a copy of this Consent form.

C.10 Declaration by the athlete

If you are willing to participate in this research project, please sign the Declaration of Consent below and return it to the principle investigator (Euodia).

I, ____ hereby voluntarily grant or deny (encircle which is applicable) my permission to be a participant in the project as explained to me by Euodia Vermeulen. The following statements apply to me, should I consent to participation:

1. I may withdraw from the study at any time and am under no obligation to finish the proposed study. The nature, objective, possible safety and health implications have been explained to me and I understand them. I have read the above information and it is written in a language with which I am fluent and comfortable.

2. I understand my right to choose whether to participate in the project is completely voluntary and that the information obtained will be handled and stored confidentially. I am aware that the results of the investigation will be used for the purposes of publication. The information will only be used for research purposes.
3. I grant the researcher access to my online fitness profile for the sake of data extraction and validation thereof.
4. I understand that I have access to all my data as well as results pertaining to me.
5. I understand that if I do not want to participate in this study I am free to continue running as I have been doing without any restrictions or prejudice from the researcher.

I have read or had read to me in a language that I understand the above information before signing this consent form. The content and meaning of this information have been explained to me. I have been given opportunity to ask questions and am satisfied that they have been answered satisfactorily. I understand that if I do not participate it will not alter my running activities in any way. I hereby volunteer to take part in this study.

Signed at (place) ______________________________

____________________ ____________________

Athlete signature Date

C.11 Biographical information, VO_2-max and injury history

Please fill in the following, if you choose to:

1. Age _______________
2. Biological sex (encircle): Male Female Choose not not disclose
3. VO_2-max: _______________
4. Height _______________
5. Injury history: please provide your injury history (more or less the date or a time frame, diagnosis and recovery time) since you started running. (Only if you are comfortable to share the diagnosis.) You may use the last page of this document.

Please promptly inform the researcher (Euodia) if you do develop an injury during the course of the study, and if possible the diagnosis (only if you are comfortable to share the diagnosis.)

Stress score, or a similar variable that represents physical readiness and/or recovery (as a function of heart rate variability and other variables measured by the device) is extracted for each day.

C.12 Declaration by the principal investigator

As the principal investigator I hereby declare that the information contained in this document has been thoroughly explained to the participant. I also declare that the participant has been encouraged (and has been given ample time) to ask any questions. In addition, I would like to select the following option:

	The conversation with the participant was conducted in a language in which the participant is fluent.
	The conversation with the participant was conducted with the assistance of a translator, and this "Consent Form" is available to the participant in a language in which the participant is fluent.

Signed at (place) ______________________

______________________ Investigator signature

______________________ Date

C.13 Injury history

Injury history: please provide your injury history (more or less the date or a time frame, diagnosis and recovery time) since you started running. (Only if you are comfortable to share the diagnosis.)

Appendix D

Simulation documentation

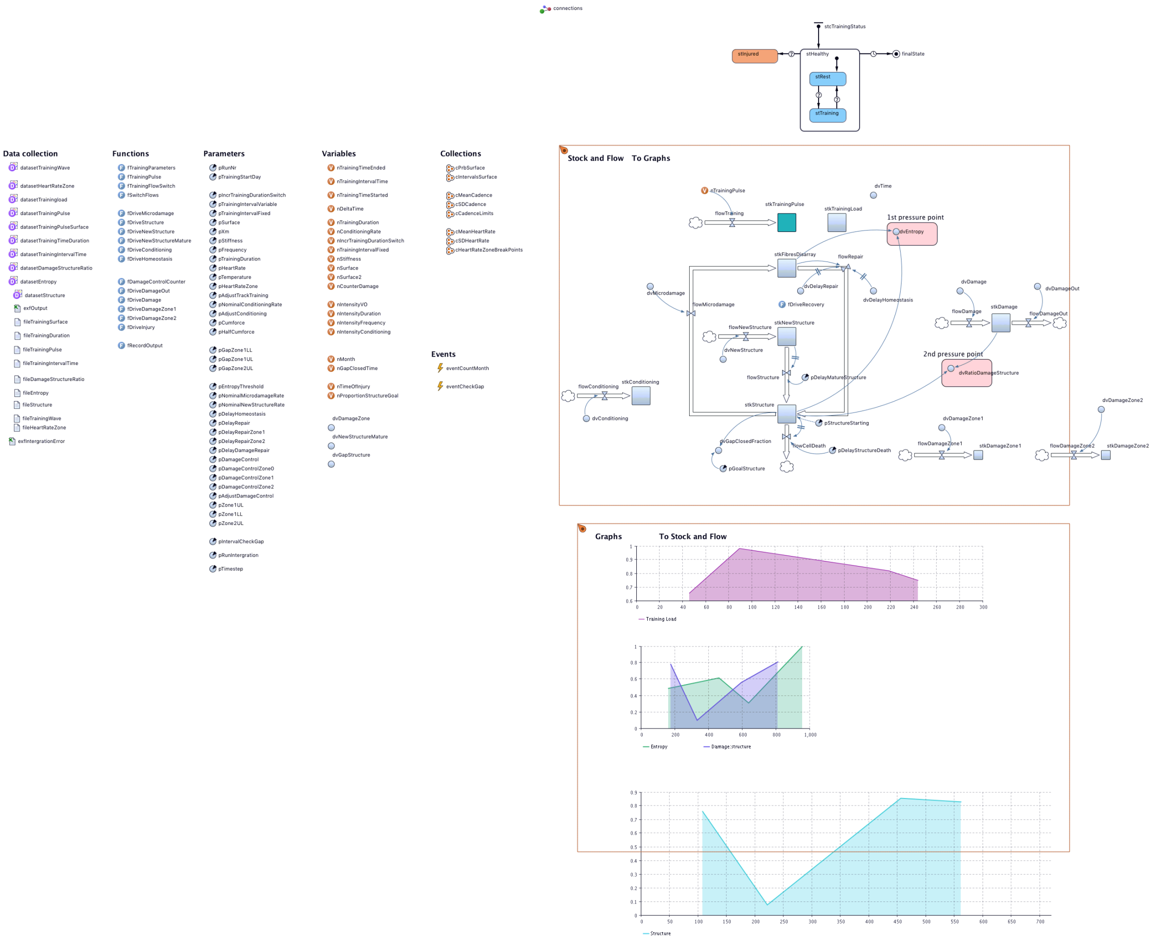
The documentation of the entire simulation model is provided here, exported from the *Anylogic* software.

Model: RCASvisual2

Name	Value
General	
Model time units	days
Numerical methods	
Differention Equations Method	Euler
Algebraic Equations Method	Modified Newton
Mixed Equations Method	RK45+Newton
Absolute accuracy	1.0E-5
Time accuracy	1.0E-5
Relative accuracy	1.0E-5
Fixed time step	0.001
Advanced	
Java package name	rcasvisual2
File Name	/Users/euodiavermeulen/Models/RCASvisual2/RCASvisual2.alp

Agent Type: Main

Name	Value
Agent in flowcharts	
Use in flowcharts as	Agent
Dimensions and movement	
Speed	(10 : MPS)
Rotate animation towards movement	true
Rotate vertically as well (along Z-axis)	false
Space and network	
Space Type	Continuous
Advanced Java	
Generic	false
Advanced	
Logging	true
Auto-create datasets	true
AOC_DATASETS_UPDATE_TIME_PROPERTIES	- Recurring Event Properties
Limit the number of data samples	false



Parameter: pXm

Description: Range of the VO (max height of the com to the min height), in meters.
Taken from Ferris (1998).

Name	Value
General	
Array	false
Default value	normal(0.08, 0.12, 0.1, 0.01)
Type	double
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	
System dynamics units	false
Save in snapshot	true
Description	
Description	Range of the VO (max height of the com to the min height), in

Name	Value
	meters. Taken from Ferris (1998).

Parameter: pFrequency

Description: cadence (steps per second)

cadence (steps per hour) = cadence (steps / min) * 60 (min / hour)

Name	Value
General	
Array	false
Default value	normal(2.5, 3.33, 3, 0.25)
Type	double
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	
System dynamics units	false
Save in snapshot	true
Description	
Description	cadence (steps per second) cadence (steps per hour) = cadence (steps / min) * 60 (min / hour)

Parameter: pStiffness

Description: stiffness coefficient of the spring (k):
mg / vo

Use values from Ferris (1998): between 17.8 and 26.6

Name	Value
General	
Array	false
Default value	20
Type	double
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	
System dynamics units	false
Save in snapshot	true
Description	
Description	stiffness coefficient of the spring (k): mg / vo Use values from Ferris (1998): between 17.8 and 26.6

Parameter: pGoalStructure

Name	Value
General	

Name	Value
Array	false
Default value	29468500.69
Type	double
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	
System dynamics units	false
Save in snapshot	true

Parameter: pDelayRepair

Name	Value
General	
Array	false
Default value	1
Type	double
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	
System dynamics units	false
Save in snapshot	true

Parameter: pDamageControl

Description: The quick-fix solution to remove small amounts of damage.

Name	Value
General	
Array	false
Default value	1
Type	double
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	
System dynamics units	false
Save in snapshot	true
Description	
Description	The quick-fix solution to remove small amounts of damage.

Parameter: pDelayDamageRepair

Description: The do-nothing number of days that the body will be able to completely repair itself, in the absence of training.

Name	Value

Name	Value
General	
Array	false
Default value	200
Type	double
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	
System dynamics units	false
Save in snapshot	true
Description	
Description	The do-nothing number of days that the body will be able to completely repair itself, in the absence of training.

Parameter: pDelayStructureDeath

Name	Value
General	
Array	false
Default value	1000
Type	double
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	
System dynamics units	false
Save in snapshot	true

Parameter: pNominalConditioningRate

Description: 1% of required initial structure

Name	Value
General	
Array	false
Default value	29468.50069
Type	double
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	
System dynamics units	false
Save in snapshot	true
Description	
Description	1% of required initial structure

Parameter: pDelayMatureStructure

Name	Value
General	
Array	false
Default value	3
Type	double
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	
System dynamics units	false
Save in snapshot	true

Parameter: pNominalMicrodamageRate

Name	Value
General	
Array	false
Default value	0.001
Type	double
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	
System dynamics units	false
Save in snapshot	true

Parameter: pNominalNewStructureRate

Name	Value
General	
Array	false
Default value	0.5
Type	double
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	
System dynamics units	false
Save in snapshot	true

Parameter: pIntervalCheckGap

Name	Value
General	
Array	false
Default value	1
Type	double

Name	Value
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	
System dynamics units	false
Save in snapshot	true

Parameter: pTrainingStartDay

Name	Value
General	
Array	false
Default value	1
Type	double
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	
System dynamics units	false
Save in snapshot	true

Parameter: pDelayHomeostasis

Name	Value
General	
Array	false
Default value	0.1
Type	double
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	
System dynamics units	false
Save in snapshot	true

Parameter: pSurface

Name	Value
General	
Array	false
Type	double
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	

Name	Value
System dynamics units	false
Save in snapshot	true

Parameter: pTemperature

Name	Value
General	
Array	false
Default value	24
Type	double
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	
System dynamics units	false
Save in snapshot	true

Parameter: pTrainingDuration

Description: Unit hours

Name	Value
General	
Array	false
Default value	1
Type	double
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	
System dynamics units	false
Save in snapshot	true
Description	
Description	Unit hours

Parameter: pCumforce

Name	Value
General	
Array	false
Type	double
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	
System dynamics units	false
Save in snapshot	true

Parameter: pTrainingIntervalVariable

Description: Switches to variable calculation of the interval time to the next training session (0 - off, 1 - on)

Name	Value
General	
Array	false
Default value	1
Type	double
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	
System dynamics units	false
Save in snapshot	true
Description	
Description	Switches to variable calculation of the interval time to the next training session (0 - off, 1 - on)

Parameter: pTrainingIntervalFixed

Description: Switches to fixed interval time to the next training session (0 - off, 1 - on)

Name	Value
General	
Array	false
Default value	0
Type	double
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	
System dynamics units	false
Save in snapshot	true
Description	
Description	Switches to fixed interval time to the next training session (0 - off, 1 - on)

Parameter: pHeartRateZone

Name	Value
General	
Array	false
Default value	1
Type	int
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	

Name	Value
System dynamics units	false
Save in snapshot	true

Parameter: pZone1UL

Name	Value
General	
Array	false
Default value	0.2
Type	double
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	
System dynamics units	false
Save in snapshot	true

Parameter: pZone1LL

Description: Starting value is 0.09 (after some emperical simulations early on in the modelling process).

Name	Value
General	
Array	false
Default value	0.09
Type	double
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	
System dynamics units	false
Save in snapshot	true
Description	
Description	Starting value is 0.09 (after some emperical simulations early on in the modelling process).

Parameter: pZone2UL

Name	Value
General	
Array	false
Default value	0.5
Type	double
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	

Name	Value
System dynamics units	false
Save in snapshot	true

Parameter: pStructureStarting

Description: 120% of initial training pulse

Name	Value
General	
Array	false
Default value	2946850.069
Type	double
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	
System dynamics units	false
Save in snapshot	true
Description	
Description	120% of initial training pulse

Parameter: pRunNr

Description: This value is to coordinate the experimental sequencing and data capturing. It does not influence the model.

Name	Value
General	
Array	false
Default value	128
Type	int
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	
System dynamics units	false
Save in snapshot	true
Description	
Description	This value is to coordinate the experimental sequencing and data capturing. It does not influence the model.

Parameter: pDelayRepairZone2

Name	Value
General	
Array	false
Default value	3
Type	double
Show at runtime	true
Show name	true

Name	Value
Value editor	
Editor control	Text
Advanced	
System dynamics units	false
Save in snapshot	true

Parameter: pDelayRepairZone1

Name	Value
General	
Array	false
Default value	2
Type	double
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	
System dynamics units	false
Save in snapshot	true

Parameter: pDamageControlZone1

Name	Value
General	
Array	false
Default value	4000
Type	double
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	
System dynamics units	false
Save in snapshot	true

Parameter: pDamageControlZone2

Name	Value
General	
Array	false
Default value	5000
Type	double
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	
System dynamics units	false

Name	Value
Save in snapshot	true

Parameter: pDamageControlZone0

Name	Value
General	
Array	false
Default value	1
Type	double
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	
System dynamics units	false
Save in snapshot	true

Parameter: pEntropyThreshold

Description: Set up as being relational to the micro-damage rate. Starts at double the nominal micro-damage rate (0.001).

Name	Value
General	
Array	false
Default value	0.002
Type	double
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	
System dynamics units	false
Save in snapshot	true
Description	
Description	Set up as being relational to the micro-damage rate. Starts at double the nominal micro-damage rate (0.001).

Parameter: pGapZone1LL

Description: = GapZone2UL

Name	Value
General	
Array	false
Default value	0.25
Type	double
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	

Name	Value
System dynamics units	false
Save in snapshot	true
Description	
Description	= GapZone2UL

Parameter: pGapZone1UL

Description: =GapZone2LL

Name	Value
General	
Array	false
Default value	0.5
Type	double
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	
System dynamics units	false
Save in snapshot	true
Description	
Description	=GapZone2LL

Parameter: pGapZone2UL

Description: =GapZone3UL

Name	Value
General	
Array	false
Default value	0.75
Type	double
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	
System dynamics units	false
Save in snapshot	true
Description	
Description	=GapZone3UL

Parameter: pIncrTrainingDurationSwitch

Name	Value
General	
Array	false
Default value	0
Type	int

Name	Value
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	
System dynamics units	false
Save in snapshot	true

Parameter: pHeartRate

Name	Value
General	
Array	false
Type	double
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	
System dynamics units	false
Save in snapshot	true

Parameter: pHalfCumforce

Name	Value
General	
Array	false
Type	double
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	
System dynamics units	false
Save in snapshot	true

Parameter: pTimestep

Name	Value
General	
Array	false
Default value	0.1
Type	double
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	
System dynamics units	false

Name	Value
Save in snapshot	true

Parameter: pRunIntergration

Name	Value
General	
Array	false
Default value	5
Type	int
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	
System dynamics units	false
Save in snapshot	true

Parameter: pAdjustTrackTraining

Description: =GapZone3UL

Name	Value
General	
Array	false
Default value	1
Type	double
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	
System dynamics units	false
Save in snapshot	true
Description	
Description	=GapZone3UL

Parameter: pAdjustConditioning

Name	Value
General	
Array	false
Default value	1
Type	double
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	
System dynamics units	false
Save in snapshot	true

Parameter: pAdjustDamageControl

Name	Value
General	
Array	false
Default value	8
Type	double
Show at runtime	true
Show name	true
Value editor	
Editor control	Text
Advanced	
System dynamics units	false
Save in snapshot	true

Function: fDriveStructure

Name	Value
General	
Return type	double
Return type:	Returns value
Show at runtime	true
Show name	true
Function body	
Body	double gapStructure; gapStructure = pGoalStructure - stkStructure; return gapStructure;
Advanced	
Static	false
Access type	default
System dynamics units	false

Function: fDriveDamage

Name	Value
General	
Return type	double
Return type:	Returns value
Show at runtime	true
Show name	true
Function body	

Body	double damage;
	
	if(dvEntropy >= pEntropyThreshold){
	damage = 0.25 * stkFibresDisarray;
	}
	else damage = 0;
	
	return damage;

Advanced	

Name	Value
Static	false
Access type	default
System dynamics units	false

Function: fDriveRecovery

Description: This function monitors the position of the athlete in the damage zones and adjust the parameters accordingly.
The path to injury is driven by increasing the repair delay between fibres in disarray and structure, adjustments in damage control and adjustments in natural cell death.
The conditioning rate, nConditioningRate, is set as a factor of the nominal rate.

What is returned by the function is the adjusted value for the delay of repair of the fibres in dissaray (dvDelayRepair).

Name	Value
General	
Return type	double
Return type:	Returns value
Show at runtime	true
Show name	true
Function body	

Body	double delay_repair;
	
	if(dvRatioDamageStructure <= pZone1LL){
	delay_repair = pDelayRepair;
	pDamageControl = pDamageControlZone0;
	pDelayStructureDeath = 1000;
	nConditioningRate = pAdjustConditioning *
	pNominalConditioningRate;
	nIncrTrainingDurationSwitch = 0;}
	
	//danger zone: the weakening of structures through increased repair delay (by a factor)
	///adjust damage control upward(the quick-fix to shift the burden)
	///more training time as the fix-that-failed
	else if(dvRatioDamageStructure > pZone1LL &&
	dvRatioDamageStructure <= pZone1UL){
	delay_repair = pDelayRepairZone1 * pDelayRepair; //weakening of structures
	pDamageControl = pAdjustDamageControl *
	pDamageControlZone1 / nCounterDamage; //the quick-fix to shift the burden
	nIncrTrainingDurationSwitch = pIncrTrainingDurationSwitch ; //more training as the fix that failed
	nConditioningRate = pAdjustConditioning *
	pNominalConditioningRate;
	pDelayStructureDeath = 800;
	}
	
	else if(dvRatioDamageStructure > pZone1UL &&
	dvRatioDamageStructure <= pZone2UL){
	delay_repair = pDelayRepairZone2 * pDelayRepair; //weakening of structures
	pDamageControl = pAdjustDamageControl *
	pDamageControlZone2 / nCounterDamage; //the quick-fix to shift the burden
	nIncrTrainingDurationSwitch = pIncrTrainingDurationSwitch; //more training: the fix that failed
	nConditioningRate = pAdjustConditioning *
	pNominalConditioningRate;
	pDelayStructureDeath = 800;}
	
	else delay_repair = pDelayRepair;
	
	return delay_repair;

Advanced	

Name	Value
Static	false
Access type	default
System dynamics units	false
Description	
Description	This function monitors the position of the athlete in the damage zones and adjust the parameters accordingly. The path to injury is driven by increasing the repair delay between fibres in disarray and structure, adjustments in damage control and adjustments in natural cell death. The conditioning rate, nConditioningRate, is set as a factor of the nominal rate. What is returned by the function is the adjusted value for the delay of repair of the fibres in dissaray (dvDelayRepair).

Function: fDriveConditioning

Description: Adjusts the intensity of the conditioning as the athlete moves through the fitness zones, based on the fraction of the closed gap. The conditioning rate (nConditioningRate) is set in the function fDriveRecovery(), as a factor of the nominal rate.

Name	Value
General	
Return type	double
Return type:	Returns value
Show at runtime	true
Show name	true
Function body	

Body	double flow;
	
	if (dvGapClosedFraction > pGapZone1LL &&
	dvGapClosedFraction <= pGapZone1UL){
	nIntensityConditioning = 1.1;
	}
	
	else if(dvGapClosedFraction > pGapZone1UL &&
	dvGapClosedFraction <= pGapZone2UL){
	nIntensityConditioning = pow(1.1, 2);
	}
	
	else if(dvGapClosedFraction >= pGapZone2UL){
	nIntensityConditioning = pow(1.1, 3);
	}
	
	else {nIntensityConditioning = 1;}
	
	//adjust the intensity
	flow = nIntensityConditioning * nConditioningRate;
	
	return flow;

Advanced	
Static	false
Access type	default
System dynamics units	false
Description	
Description	Adjusts the intensity of the conditioning as the athlete moves through the fitness zones, based on the fraction of the closed gap. The conditioning rate (nConditioningRate) is set in the function fDriveRecovery(), as a factor of the nominal rate.

Function: fDriveDamageOut

Description: The outflow remains at 0 when there is no content in the stock for damage.
Otherwise, the outflow is a function of the parameter pDamageControl, which is dynamic and set in fDriveRecovery().
There is also a natural damage repair delay, which the body does irrespective of additional steps taken by the athlete.

Name	Value
General	
Return type	double
Return type:	Returns value
Show at runtime	true
Show name	true
Function body	

Body	double outflow;
	
	if(stkDamage <= 0){
	outflow = 0;
	}
	else
	outflow = pDamageControl + stkDamage / pDelayDamageRepair;
	
	return outflow;

Advanced	
Static	false
Access type	default
System dynamics units	false
Description	
Description	The outflow remains at 0 when there is no content in the stock for damage. Otherwise, the outflow is a function of the parameter pDamageControl, which is dynamic and set in fDriveRecovery(). There is also a natural damage repair delay, which the body does irrespective of additional steps taken by the athlete.

Function: fDriveMicrodamage

Description: As the gap closes: the value for (dvGap / pGoal) becomes smaller, so entire flow becomes slower.
The function switches to a nominal rate when the gap is very close to being closed or breached, this is in order to prevent a negative flow.

Name	Value
General	
Return type	double
Return type:	Returns value
Show at runtime	true
Show name	true
Function body	

Body	double m;
	
	if (flowTraining == 0){
	m = 0;
	}
	
	else {
	//slow down the micro damage rate as structure closes in on goal
	if(dvGapClosedFraction <= 0.995){
	m = ((dvGapStructure / pGoalStructure) * pNominalMicrodamageRate * flowTraining) / 0.0417;
	}
	
	//maintain a constant nominal rate for micro-damage when the gap has been closed or is very small
	else { m = 0.005 * pNominalMicrodamageRate * flowTraining / 0.0417;
	}
	
	}

Name	Value
	return m;
Advanced	
Static	false
Access type	default
System dynamics units	false
Description	
Description	As the gap closes: the value for (dvGap / pGoal) becomes smaller, so entire flow becomes slower. The function switches to a nominal rate when the gap is very close to being closed or breached, this is in order to prevent a negative flow.

Function: fDriveNewStructure

Name	Value
General	
Return type	double
Return type:	Returns value
Show at runtime	true
Show name	true
Function body	

Body	double flow;
	
	//flow as a function of conditioning and micro-damage from training
	
	flow = 0.75 * flowConditioning + pNominalNewStructureRate * flowMicrodamage;
	
	return flow;

Advanced	
Static	false
Access type	default
System dynamics units	false

Function: fTrainingParameters

Description: Set-up the training parameters and the biomechanical metrics for the run session.
Use Ferris (1997) to link leg stiffness, VO and surface parameters.

Sample temperature values.
Assign the sampled heart rate to a heart rate zone.
Temperature and heart rate zones are to be used in the drive of homeostasis (fDriveHomeostasis).
Determine intensity as a function of the gap and adjust the conditioning and training duration accordingly.

Name	Value
General	
Return type:	Just action (returns nothing)
Show at runtime	true
Show name	true
Function body	

Body	//set parameters
	//get random uniform number to sample from the surface type pmf
	nSurface = uniform();
	
	//find the interval for the random number and assign surface type
	//1 - rc, 2 - rr, 3 - tc, 4 - tr, 5 - tt
	if(nSurface < cIntervalsSurface.get(0)){
	nSurface2 = 1;}
	else if(nSurface >= cIntervalsSurface.get(0) && nSurface <

Name	Value

	cIntervalsSurface.get(1)){
	nSurface2 = 2; }
	else if(nSurface >= cIntervalsSurface.get(1) && nSurface <
	cIntervalsSurface.get(2)){
	nSurface2 = 3;}
	else if(nSurface >= cIntervalsSurface.get(2) && nSurface <
	cIntervalsSurface.get(3)){
	nSurface2 = 4;}
	else if(nSurface >= cIntervalsSurface.get(3)){
	nSurface2 = 5;}
	else nSurface2 = 0;
	
	//set environmental parameters
	//temperature has an influence on the homeostasis delay
	if(nMonth == 1 | nMonth == 13){
	pTemperature = normal(14.32, 30.86, 22.98, 4.407);
	}
	else if(nMonth == 2 | nMonth == 14){
	pTemperature = normal(12.81, 31.83, 22.69 , 5.327);
	}
	else if(nMonth == 3 | nMonth == 15){
	pTemperature = normal(9.51 , 28.67 , 20.13 , 5.327);
	}
	else if(nMonth == 4 | nMonth == 16){
	pTemperature = normal(8.33 , 27.810 , 18.17 , 4.594);
	}
	else if(nMonth == 5 | nMonth == 17){
	pTemperature = normal(0.7103, 23.33, 14.636, 5.32);
	}
	else if(nMonth == 6 | nMonth == 18){
	pTemperature = normal(3.73, 22.93, 12.77, 5.024);
	}
	else if(nMonth == 7 | nMonth == 19){
	pTemperature = normal(0.86, 25.10, 13.32, 6.343);
	}
	else if(nMonth == 8 | nMonth == 20){
	pTemperature = normal(7.39, 27.31 , 17.22, 5.909);
	}
	else if(nMonth == 9 | nMonth == 21){
	pTemperature = normal(2.71 , 31.70, 18.89, 6.776);
	}
	else if(nMonth == 10 | nMonth == 22){
	pTemperature = normal(7.49, 34.69 , 22.88 , 6.408);
	}
	else if(nMonth == 11 | nMonth == 23){
	pTemperature = normal(13.16, 29.62 , 21.87, 4.314);
	}
	else if(nMonth == 12 | nMonth == 24){
	pTemperature = normal(12.47, 33.08 , 22.03, 5.358);
	}
	else pTemperature = 25;
	
	//adjust training parameters to surface
	if(nSurface2 == 1){
	pFrequency = normal(cMeanCadence.get(0) - 3 *
	cSDCadence.get(0), cMeanCadence.get(0) + 3 *
	cSDCadence.get(0),
	cMeanCadence.get(0), cSDCadence.get(0)) ;
	nStiffness = pStiffness;
	pXm = normal(0.1, 0.15, 0.12, 0.012);
	//pHeartRate = normal(cMeanHeartRate.get(0) - 3 *
	cSDHeartRate.get(0), cMeanHeartRate.get(0) + 3 *
	cSDHeartRate.get(0),
	//cMeanHeartRate.get(0), cSDHeartRate.get(0));
	pSurface = nSurface2;
	pAdjustTrackTraining = 1;
	}
	else if(nSurface2 == 2){
	pFrequency = normal(cMeanCadence.get(1) - 3 *
	cSDCadence.get(1), cMeanCadence.get(1) + 3 *
	cSDCadence.get(1),
	cMeanCadence.get(1), cSDCadence.get(1)) ;

Name	Value

	//pHeartRate = normal(cMeanHeartRate.get(1) - 3 * cSDHeartRate.get(1), cMeanHeartRate.get(1) + 3 * cSDHeartRate.get(1),
	//cMeanHeartRate.get(1), cSDHeartRate.get(1));
	pSurface = nSurface2;
	pAdjustTrackTraining = 1;
	}
	else if(nSurface2 == 3){
	pFrequency = normal(cMeanCadence.get(2) - 3 * cSDCadence.get(2), cMeanCadence.get(2) + 3 * cSDCadence.get(2),
	cMeanCadence.get(2), cSDCadence.get(2)) ;
	nStiffness = 1.5 * pStiffness;
	pXm = normal(0.067, 0.1, 0.08, 0.008);
	//pHeartRate = normal(cMeanHeartRate.get(2) - 3 * cSDHeartRate.get(2), cMeanHeartRate.get(2) + 3 * cSDHeartRate.get(2),
	//cMeanHeartRate.get(2), cSDHeartRate.get(2));
	pSurface = nSurface2;
	pAdjustTrackTraining = 1;
	}
	else if(nSurface2 == 4){
	pFrequency = normal(cMeanCadence.get(3) - 3 * cSDCadence.get(3), cMeanCadence.get(3) + 3 * cSDCadence.get(3),
	cMeanCadence.get(3), cSDCadence.get(3)) ;
	nStiffness = 1.5 * pStiffness;
	pXm = normal(0.067, 0.1, 0.08, 0.008);
	//pHeartRate = normal(cMeanHeartRate.get(3) - 3 * cSDHeartRate.get(3), cMeanHeartRate.get(3) + 3 * cSDHeartRate.get(3),
	//cMeanHeartRate.get(3), cSDHeartRate.get(3));
	pSurface = nSurface2;
	pAdjustTrackTraining = 1;
	}
	else if(nSurface2 == 5){
	//pFrequency = normal(cMeanCadence.get(4) - 3 * cSDCadence.get(4), cMeanCadence.get(4) + 3 * cSDCadence.get(4),
	//cMeanCadence.get(4), cSDCadence.get(4)) ;
	nStiffness = 2 * pStiffness;
	pXm = normal(0.05, 0.075, 0.06, 0.006);
	//pHeartRate = normal(cMeanHeartRate.get(4) - 3 * cSDHeartRate.get(4), cMeanHeartRate.get(4) + 3 * cSDHeartRate.get(4),
	//cMeanHeartRate.get(4), cSDHeartRate.get(4));
	pSurface = nSurface2;
	pAdjustTrackTraining = 1;
	if(randomTrue(0.5)){
	pFrequency = normal(cMeanCadence.get(4) - 3 * cSDCadence.get(4), cMeanCadence.get(4) + 3 * cSDCadence.get(4),
	cMeanCadence.get(4), cSDCadence.get(4));
	pAdjustTrackTraining = 0.53;}
	else {pFrequency = normal(cMeanCadence.get(5) - 3 * cSDCadence.get(5), cMeanCadence.get(5) + 3 * cSDCadence.get(5),
	cMeanCadence.get(5), cSDCadence.get(5));
	pAdjustTrackTraining = 1;}
	
	}
	
	//cadence - heart rate interaction
	if(pFrequency < cCadenceLimits.get(0)){
	if(randomTrue(0.668)){
	pHeartRate = normal(116, 145, 134, 7.508); }
	else pHeartRate = normal(146, 172, 156.44, 6.779); }
	else if(pFrequency < cCadenceLimits.get(1) && pFrequency >= cCadenceLimits.get(0)){
	if(randomTrue(0.3898)){
	pHeartRate = normal(116, 145, 134, 7.508); }
	else pHeartRate = normal(146, 172, 156.44, 6.779); }
	else if(pFrequency >= cCadenceLimits.get(1)){
	if(randomTrue(0.3216)){

Name	Value

	//{117.91, 136.05, 145.12, 154.19, 163.26}
	if(pHeartRate <= cHeartRateZoneBreakPoints.get(0)){
	pHeartRateZone = 1;
	}
	else if(pHeartRate > cHeartRateZoneBreakPoints.get(0) &&
	pHeartRate <= cHeartRateZoneBreakPoints.get(1)){
	pHeartRateZone = 2;
	}
	else if(pHeartRate > cHeartRateZoneBreakPoints.get(1) &&
	pHeartRate <= cHeartRateZoneBreakPoints.get(2)){
	pHeartRateZone = 3;
	}
	else if(pHeartRate > cHeartRateZoneBreakPoints.get(2) &&
	pHeartRate <= cHeartRateZoneBreakPoints.get(3)){
	pHeartRateZone = 4;
	}
	else if(pHeartRate > cHeartRateZoneBreakPoints.get(3)){
	pHeartRateZone = 5;
	}
	
	//pHeartRateZone = cHeartRateZone.get(uniform_discr(0,4));
	
	// set the intensity of training, based on the size of the gap
	//determine the intensity of cadence and vo as a function of the gap
	//set the parameters to 1 if the intensity should remain the same
	if (dvGapClosedFraction > pGapZone1LL &&
	dvGapClosedFraction <= pGapZone1UL){
	//nIntensityFrequency = 1.05;
	//nIntensityVO = 1.1;
	nIntensityConditioning = 1.1;
	nIntensityDuration = 1.1;
	}
	
	else if(dvGapClosedFraction > pGapZone1UL &&
	dvGapClosedFraction <= pGapZone2UL){
	//nIntensityFrequency = pow(1.05, 2);
	//nIntensityVO = 1.2;
	nIntensityConditioning = pow(1.1, 2);
	nIntensityDuration = pow(1.1, 2);
	}
	
	else if(dvGapClosedFraction >= pGapZone2UL){
	//nIntensityFrequency = pow(1.05, 3);
	//nIntensityVO = 1.3;
	nIntensityConditioning = pow(1.1, 3);
	nIntensityDuration = pow(1.1, 3);
	}
	
	else {nIntensityFrequency = 1;
	nIntensityDuration = 1;}
	
	//increase training time:
	//(normal * ramp-up) + (the fix-that-failed : 'more training to fix poor fitness')
	//nTrainingDuration = pTrainingDuration * 0.0417 +
	nIncrTrainingDurationSwitch * 0.01;
	//nTrainingDuration = nIntensityDuration * pTrainingDuration *
	0.0417 * pAdjustTrackTraining;
	nTrainingDuration = pTrainingDuration * 0.0417 *
	pAdjustTrackTraining + nIncrTrainingDurationSwitch * 0.01;

Advanced	
Static	false
Access type	default

Name	Value
System dynamics units	false
Description	
Description	Set-up the training parameters and the biomechanical metrics for the run session. Use Ferris (1997) to link leg stiffness, VO and surface parameters. Sample temperature values. Assign the sampled heart rate to a heart rate zone. Temperature and heart rate zones are to be used in the drive of homeostasis (fDriveHomeostasis). Determine intensity as a function of the gap and adjust the conditioning and training duration accordingly.

Function: fTrainingFlowSwitch

Description: Training is a pulsed input and the flow should only be switched on for a part of the day for the duration of the training session

Name	Value
General	
Return type	double
Return type:	Returns value
Show at runtime	true
Show name	true
Function body	

Body	//training as a pulsed input: switch training on and off
	double training_flow;
	
	if(time() < pTrainingStartDay){
	training_flow = 0;}
	else if (time() >= pTrainingStartDay && time() <=
	pTrainingStartDay + nTrainingDuration){
	training_flow = 1;
	}
	else if (time() >= nTrainingTimeEnded + nTrainingIntervalTime
	&& time() <= (nTrainingTimeEnded + nTrainingIntervalTime +
	nTrainingDuration)){
	training_flow = 1;
	}
	else training_flow = 0;
	
	return training_flow;

Advanced	
Static	false
Access type	default
System dynamics units	false
Description	
Description	Training is a pulsed input and the flow should only be switched on for a part of the day for the duration of the training session

Function: fDriveHomeostasis

Description: Warmer temperatures and higher heart rate zones slow down homeostasis by increasing the delay.

Name	Value
General	
Return type	double
Return type:	Returns value
Show at runtime	true
Show name	true
Function body	

Name	Value

Body	double delayHomeostasis;
	
	//pDelayHomeostasis = 0.005 * pTemperature + 0.02 * pHeartRateZone;
	//delayHomeostasis = fTrainingFlowSwitch() * stkFibresDisarray / pDelayHomeostasis;
	
	delayHomeostasis = pTrainingDuration * (0.005 * pTemperature + 0.02 * pHeartRateZone);
	
	return delayHomeostasis;

Advanced	
Static	false
Access type	default
System dynamics units	false
Description	
Description	Warmer temperatures and higher heart rate zones slow down homeostasis by increasing the delay.

Function: fDriveDamageZone1

Description: Activate the flow to count the days the athlete spends in damage zone 1.

Name	Value
General	
Return type	double
Return type:	Returns value
Show at runtime	true
Show name	true
Function body	

Body	double flow;
	
	if(dvRatioDamageStructure > pZone1LL &&
	dvRatioDamageStructure < pZone1UL){
	flow = 1;
	}
	else flow = 0;
	
	return flow;

Advanced	
Static	false
Access type	default
System dynamics units	false
Description	
Description	Activate the flow to count the days the athlete spends in damage zone 1.

Function: fDriveDamageZone2

Description: Activate the flow to count the days the athlete spends in damage zone 2.

Name	Value
General	
Return type	double
Return type:	Returns value
Show at runtime	true

Name	Value
Show name	true
Function body	

Body	double flow;
	
	if(dvRatioDamageStructure >= pZone1UL){
	flow = 1;
	}
	
	else flow = 0;
	
	return flow;

Advanced	
Static	false
Access type	default
System dynamics units	false
Description	
Description	Activate the flow to count the days the athlete spends in damage zone 2.

Function: fSwitchFlows

Description: Stops all the flows in the simulation model when the athlete has transitioned out of the healthy state.
That is, all flows come to a halt when wither the athlete has been injured or the simulation time of 720 days has elapsed.

Name	Value
General	
Return type	double
Return type:	Returns value
Show at runtime	true
Show name	true
Function body	

Body	double m;
	
	if(inState(stInjured)){
	m = 0;}
	
	else if(inState(finalState)){
	m = 0;
	}
	
	else m = 1;
	
	return m;

Advanced	
Static	false
Access type	default
System dynamics units	false
Description	
Description	Stops all the flows in the simulation model when the athlete has transitioned out of the healthy state. That is, all flows come to a halt when wither the athlete has been injured or the simulation time of 720 days has elapsed.

Function: fRecordOutput

Description: Record the relevant output to either the Excel file or respective text files.
The values for the intergration errors are only recorded once when the integration error and time steps are tested.

Name	Value
General	
Return type:	Just action (returns nothing)
Show at runtime	true
Show name	true
Function body	

Body	nTimeOfInjury = time();
	//nProportionStructureGoal = (pGoalStructure - dvGapStructure) / pGoalStructure;
	double damage_cntr_z1 = pAdjustDamageControl * pDamageControlZone1;
	double damage_cntr_z2 = pAdjustDamageControl * pDamageControlZone2;
	
	//record relevant metrics and output to excel file
	exfOutput.setCellValue(pRunNr, 1, pRunNr + 1, 1); //simulation run nr
	exfOutput.setCellValue(pGoalStructure, 1, pRunNr + 1, 2);
	exfOutput.setCellValue(dvGapStructure, 1, pRunNr + 1, 3);
	exfOutput.setCellValue(dvGapClosedFraction, 1, pRunNr + 1, 4);
	exfOutput.setCellValue(nGapClosedTime, 1, pRunNr + 1, 5);
	exfOutput.setCellValue(nTimeOfInjury, 1, pRunNr + 1, 6);
	exfOutput.setCellValue(stkTrainingLoad, 1, pRunNr + 1, 7);
	exfOutput.setCellValue(stkDamageZone1, 1, pRunNr + 1, 8);
	exfOutput.setCellValue(stkDamageZone2, 1, pRunNr + 1, 9);
	exfOutput.setCellValue(pDelayRepair, 1, pRunNr + 1, 10);
	exfOutput.setCellValue(pDelayRepairZone1, 1, pRunNr + 1, 11);
	exfOutput.setCellValue(pDelayRepairZone2, 1, pRunNr + 1, 12);
	exfOutput.setCellValue(damage_cntr_z1, 1, pRunNr + 1, 13);
	exfOutput.setCellValue(damage_cntr_z2, 1, pRunNr + 1, 14);
	exfOutput.setCellValue(pDamageControlZone0, 1, pRunNr + 1, 15);
	exfOutput.setCellValue(pNominalMicrodamageRate, 1, pRunNr + 1, 16);
	exfOutput.setCellValue(pNominalNewStructureRate, 1, pRunNr + 1, 17);
	exfOutput.setCellValue(nConditioningRate, 1, pRunNr + 1, 18);
	exfOutput.setCellValue(pTrainingDuration, 1, pRunNr + 1, 19);
	exfOutput.setCellValue(nIntensityDuration, 1, pRunNr + 1, 20);
	exfOutput.setCellValue(nIntensityFrequency, 1, pRunNr + 1, 21);
	exfOutput.setCellValue(nIntensityVO, 1, pRunNr + 1, 22);
	exfOutput.setCellValue(nIntensityConditioning, 1, pRunNr + 1, 23);
	exfOutput.setCellValue(pEntropyThreshold, 1, pRunNr + 1, 24);
	exfOutput.setCellValue("variable", 1, pRunNr + 1, 25);
	exfOutput.setCellValue(nTrainingIntervalTime, 1, pRunNr + 1, 26);
	// placeholder for the mean of variable training intervals
	exfOutput.setCellValue(pStructureStarting, 1, pRunNr + 1, 27);
	exfOutput.setCellValue(pGapZone1LL, 1, pRunNr + 1, 28);
	exfOutput.setCellValue(pGapZone1UL, 1, pRunNr + 1, 29);
	exfOutput.setCellValue(pGapZone2UL, 1, pRunNr + 1, 30);
	exfOutput.setCellValue(pDelayMatureStructure, 1, pRunNr + 1, 31);
	exfOutput.setCellValue(pIncrTrainingDurationSwitch, 1, pRunNr + 1, 32);
	exfOutput.setCellValue(pZone1LL, 1, pRunNr + 1, 33);
	exfOutput.setCellValue(pZone1UL, 1, pRunNr + 1, 34);
	exfOutput.setCellValue(pZone2UL, 1, pRunNr + 1, 35);
	exfOutput.setCellValue("", 1, pRunNr + 1, 36); //experiment name
	//exfOutput.setCellValue(pStructureStarting, 1, pRunNr + 1, 37);
	//exfOutput.setCellValue(pEntropyThreshold, 1, pRunNr + 1, 38);
	
	fileTrainingPulse.println(datasetTrainingPulse);
	fileTrainingSurface.println(datasetTrainingPulseSurface);
	fileTrainingDuration.println(datasetTrainingTimeDuration);
	fileTrainingIntervalTime.println(datasetTrainingIntervalTime);
	fileDamageStructureRatio.println(datasetDamageStructureRatio);
	fileEntropy.println(datasetEntropy);
	fileTrainingWave.println(datasetTrainingWave);
	fileHeartRateZone.println(datasetHeartRateZone);
	
	//exfIntergrationError.setCellValue(pRunIntergration, 1, pRunIntergration + 1, 1);

Name	Value

	//exfIntergrationError.setCellValue(pTimestep, 1, pRunIntergration + 1, 2);
	//exfIntergrationError.setCellValue(stkStructure, 1, pRunIntergration + 1, 3);
	//exfIntergrationError.setCellValue(stkTrainingLoad, 1, pRunIntergration + 1, 4);

Advanced	
Static	false
Access type	default
System dynamics units	false
Description	
Description	Record the relevant output to either the Excel file or respective text files. The values for the intergration errors are only recorded once when the integration error and time steps are tested.

Function: fDriveNewStructureMature

Name	Value
General	
Return type	double
Return type:	Returns value
Show at runtime	true
Show name	true
Function body	
Body	double flow; flow = fSwitchFlows() * stkNewStructure/ pDelayMatureStructure; return flow;
Advanced	
Static	false
Access type	default
System dynamics units	false

Function: fTrainingPulse

Description: The function calculates the integral of the cosine waveform that represents the variable force of the spring-mass system.
The output is the total impulse in kN-s for the training session.

The parameters in the for-loop have been set-up in fTrainingParameters().

From Cavagna (2008): take, at first, a perfect symmetry for the SHM and count 50% as stance phase (Sc).
For future experiments, this value may be adjusted.

Name	Value
General	
Return type:	Just action (returns nothing)
Show at runtime	true
Show name	true
Function body	

Body	double t = 0;
	double cumforce = 0;
	
	nTrainingPulse = 0;
	pCumforce = 0;

Name	Value

	//generate a training pulse for one hour
	for(int i = 1; i <= 3600000; i++){
	//nCounter ++;
	cumforce += abs((- nStiffness) * pXm
	* cos(pFrequency * 2*PI * t));
	datasetTrainingWave.add(t, (- nStiffness) * pXm
	* cos(pFrequency * 2*PI * t));
	t += 0.001;
	}
	
	pCumforce = cumforce;
	pHalfCumforce = (0.5 * cumforce);
	//pTrainingPulse = (0.5 * cumforce) / 0.0417;
	
	//divide by (1/24 = 0.0417) to amplify the pulse so that the hour's worth of training will be pulsed
	nTrainingPulse = (0.5 * cumforce) / 0.0417;

Advanced	
Static	false
Access type	default
System dynamics units	false
Description	
Description	The function calculates the integral of the cosine waveform that represents the variable force of the spring-mass system. The output is the total impulse in kN-s for the training session. The parameters in the for-loop have been set-up in fTrainingParameters(). From Cavagna (2008): take, at first, a perfect symmetry for the SHM and count 50% as stance phase (Sc). For future experiments, this value may be adjusted.

Function: fDamageControlCounter

Description: The denominator to adjust the efficacy of the damage control mechanism is updated.
The efficacy of the damage control action decresases hyperbolically (inversely proportional to the time it has been used).

Name	Value
General	
Return type:	Just action (returns nothing)
Show at runtime	true
Show name	true
Function body	

Body	
	if(dvRatioDamageStructure <= pZone1LL){
	nCounterDamage = 1;
	}
	
	else if(dvRatioDamageStructure > pZone1LL &&
	dvRatioDamageStructure <= pZone1UL){
	nCounterDamage += 0.01;
	}
	
	else if(dvRatioDamageStructure > pZone1UL &&
	dvRatioDamageStructure <= pZone2UL){
	nCounterDamage += 0.1;}
	
	else nCounterDamage = 1;

Name	Value
Advanced	
Static	false
Access type	default
System dynamics units	false
Description	
Description	The denominator to adjust the efficacy of the damage control mechanism is updated. The efficacy of the damage control action decresases hyperbolically (inversely proportional to the time it has been used).

Function: fDriveInjury

Description: Monitor the time spent in the damage zone and incite the injury when the limits have been reached.
The value that is returned is only an indicator to the observer during model run time, it has no infuence on the model.

Name	Value
General	
Return type	double
Return type:	Returns value
Show at runtime	true
Show name	true
Function body	

Body	//incite the injury, when cumulative damage over time has reached a limit within the damage zones
	double r = 0;
	
	if(dvRatioDamageStructure > pZone1LL &&
	dvRatioDamageStructure < pZone1UL){
	r = 1;
	if(stkDamageZone1 >= 42){
	pDelayStructureDeath = 1;
	r = 11;
	}
	}
	else if(dvRatioDamageStructure >= pZone1UL &&
	dvRatioDamageStructure < pZone2UL){
	r = 2;
	//check whether the transition from Zone 1 is counterintuitive:
	//if the time remaining in zone 1 is less than the days allowed in zone 2, incite the injury
	if(42 - stkDamageZone1 <= 10){
	pDelayStructureDeath = 1;
	r = 21;
	}
	else if(stkDamageZone2 >= 10){
	pDelayStructureDeath = 1;
	r = 22;
	}
	}
	else r = 0;
	
	return r;

Advanced	
Static	false
Access type	default
System dynamics units	false
Description	
Description	Monitor the time spent in the damage zone and incite the injury when the limits have been reached. The value that is returned is only an indicator to the observer during model run time, it has no infuence on the model.

Event: eventCheckGap

Description: This event must capture the time at which the fitness gap was closed (or very nearly closed).
It must only capture this time once. The follow-up check time is then set to an unreachable high number, so the simulation will never reach this point.

Name	Value
General	
Logging	true
EVENT_TIMEOUT_PROPERTIES	- Recurring Event Properties
Mode	Cyclic
Trigger type	Timeout
Show at runtime	true
Show name	true
Action	

Action	if(dvGapStructure / pGoalStructure <= 0.005){
	nGapClosedTime = time();
	pIntervalCheckGap = 10000;
	}

Description	
Description	This event must capture the time at which the fitness gap was closed (or very nearly closed). It must only capture this time once. The follow-up check time is then set to an unreachable high number, so the simulation will never reach this point.

Event: eventCountMonth

Name	Value
General	
Logging	true
EVENT_TIMEOUT_PROPERTIES	- Recurring Event Properties
Mode	Cyclic
Trigger type	Timeout
Show at runtime	true
Show name	true
Action	
Action	nMonth ++;

Variable: nTrainingDuration

Name	Value
General	
Initial value	0.084
Type	double
Show at runtime	true
Show name	true
Advanced	
Access type	public
Static	false
Constant	false
Save in snapshot	true
System dynamics units	false

Variable: nTrainingTimeEnded

Name	Value
General	
Type	double
Show at runtime	true
Show name	true
Advanced	
Access type	public
Static	false
Constant	false
Save in snapshot	true
System dynamics units	false

Variable: nTrainingIntervalTime

Name	Value
General	
Initial value	0.958
Type	double
Show at runtime	true
Show name	true
Advanced	
Access type	public
Static	false
Constant	false
Save in snapshot	true
System dynamics units	false

Variable: nTrainingTimeStarted

Name	Value
General	
Type	double
Show at runtime	true
Show name	true
Advanced	
Access type	public
Static	false
Constant	false
Save in snapshot	true
System dynamics units	false

Variable: nDeltaTime

Name	Value
General	
Type	double
Show at runtime	true
Show name	true
Advanced	

Name	Value
Access type	public
Static	false
Constant	false
Save in snapshot	true
System dynamics units	false

Variable: nIntensityFrequency

Name	Value
General	
Initial value	1
Type	double
Show at runtime	true
Show name	true
Advanced	
Access type	public
Static	false
Constant	false
Save in snapshot	true
System dynamics units	false

Variable: nIntensityDuration

Name	Value
General	
Initial value	1
Type	double
Show at runtime	true
Show name	true
Advanced	
Access type	public
Static	false
Constant	false
Save in snapshot	true
System dynamics units	false

Variable: nTrainingIntervalFixed

Name	Value
General	
Initial value	1.958
Type	double
Show at runtime	true
Show name	true
Advanced	
Access type	public
Static	false
Constant	false
Save in snapshot	true

Name	Value
System dynamics units	false

Variable: nTimeOfInjury

Name	Value
General	
Type	double
Show at runtime	true
Show name	true
Advanced	
Access type	public
Static	false
Constant	false
Save in snapshot	true
System dynamics units	false

Variable: nIntensityConditioning

Name	Value
General	
Initial value	1
Type	double
Show at runtime	true
Show name	true
Advanced	
Access type	public
Static	false
Constant	false
Save in snapshot	true
System dynamics units	false

Variable: nGapClosedTime

Name	Value
General	
Type	double
Show at runtime	true
Show name	true
Advanced	
Access type	public
Static	false
Constant	false
Save in snapshot	true
System dynamics units	false

Variable: nProportionStructureGoal

Name	Value
General	

Name	Value
Type	double
Show at runtime	true
Show name	true
Advanced	
Access type	public
Static	false
Constant	false
Save in snapshot	true
System dynamics units	false

Variable: nSurface

Name	Value
General	
Type	double
Show at runtime	true
Show name	true
Advanced	
Access type	public
Static	false
Constant	false
Save in snapshot	true
System dynamics units	false

Variable: nSurface2

Name	Value
General	
Type	double
Show at runtime	true
Show name	true
Advanced	
Access type	public
Static	false
Constant	false
Save in snapshot	true
System dynamics units	false

Variable: nMonth

Name	Value
General	
Initial value	1
Type	int
Show at runtime	true
Show name	true
Advanced	
Access type	public
Static	false

Name	Value
Constant	false
Save in snapshot	true
System dynamics units	false

Variable: nCounterDamage

Name	Value
General	
Initial value	1
Type	double
Show at runtime	true
Show name	true
Advanced	
Access type	public
Static	false
Constant	false
Save in snapshot	true
System dynamics units	false

Variable: nTrainingPulse

Name	Value
General	
Type	double
Show at runtime	true
Show name	true
Advanced	
Access type	public
Static	false
Constant	false
Save in snapshot	true
System dynamics units	false

Variable: nIncrTrainingDurationSwitch

Name	Value
General	
Type	int
Show at runtime	true
Show name	true
Advanced	
Access type	public
Static	false
Constant	false
Save in snapshot	true
System dynamics units	false

Variable: nStiffness

Name	Value
General	
Type	double
Show at runtime	true
Show name	true
Advanced	
Access type	public
Static	false
Constant	false
Save in snapshot	true
System dynamics units	false

Variable: nConditioningRate

Name	Value
General	
Initial value	pNominalConditioningRate
Type	double
Show at runtime	true
Show name	true
Advanced	
Access type	public
Static	false
Constant	false
Save in snapshot	true
System dynamics units	false

Variable: nIntensityVO

Name	Value
General	
Initial value	1
Type	double
Show at runtime	true
Show name	true
Advanced	
Access type	public
Static	false
Constant	false
Save in snapshot	true
System dynamics units	false

Collection: cPrbSurface

Description: Probability mass function generated in the script: simulation input.R
{rc, rr, tc, tr, tt}

From data analyses in R:
0.0030211 0.3987915 0.0060423 0.1540785 0.4380665

Name	Value
General	

Name	Value
Initial contents	{0.003021, 0.398792, 0.006042, 0.154079, 0.438066}
Initial contents	{0.003021, 0.398792, 0.006042, 0.154079, 0.438066}
Element class	Double
Collection class	ArrayList
Show at runtime	true
Show name	true
Advanced	
Access type	public
Save in snapshot	true
Static	false
Description	
Description	Probability mass function generated in the script: simulation input.R {rc, rr, tc, tr, tt} From data analyses in R: 0.0030211 0.3987915 0.0060423 0.1540785 0.4380665

Collection: cIntervalsSurface

Name	Value
General	
Initial contents	{}
Initial contents	{}
Element class	Double
Collection class	ArrayList
Show at runtime	true
Show name	true
Advanced	
Access type	public
Save in snapshot	true
Static	false

Collection: cHeartRateZoneBreakPoints

Description: From the breakpoints for hr zones: 117.91 136.05 145.12 154.19 163.26

Name	Value
General	
Initial contents	{117.91, 136.05, 145.12, 154.19, 163.26}
Initial contents	{117.91, 136.05, 145.12, 154.19, 163.26}
Element class	Double
Collection class	ArrayList
Show at runtime	true
Show name	true
Advanced	
Access type	public
Save in snapshot	true
Static	false
Description	
Description	From the breakpoints for hr zones: 117.91 136.05 145.12 154.19 163.26

Collection: cMeanHeartRate

Description: Order: rc, rr, tc, tr, tt

Name	Value
General	
Initial contents	{153.61, 142.65, 156.61, 148.36, 144.35 }
Initial contents	{153.61, 142.65, 156.61, 148.36, 144.35 }
Element class	Double
Collection class	ArrayList
Show at runtime	true
Show name	true
Advanced	
Access type	public
Save in snapshot	true
Static	false
Description	
Description	Order: rc, rr, tc, tr, tt

Collection: cMeanCadence

Description: Order: rc, rr, tc, tr, tt-1 , tt-2

From data analyses in R:
3.0385 2.9806 3.0753 2.9531 3.2443 2.9255

Name	Value
General	
Initial contents	{3.0385, 2.9806, 3.0753, 2.9531, 3.2443, 2.9255 }
Initial contents	{3.0385, 2.9806, 3.0753, 2.9531, 3.2443, 2.9255 }
Element class	Double
Collection class	ArrayList
Show at runtime	true
Show name	true
Advanced	
Access type	public
Save in snapshot	true
Static	false
Description	
Description	Order: rc, rr, tc, tr, tt-1 , tt-2 From data analyses in R: 3.0385 2.9806 3.0753 2.9531 3.2443 2.9255

Collection: cSDHeartRate

Description: Order: rc, rr, tc, tr, tt

Name	Value
General	
Initial contents	{9.6039, 12.3322 , 9.1253 , 13.2972, 14.1903}
Initial contents	{9.6039, 12.3322 , 9.1253 , 13.2972, 14.1903}
Element class	Double
Collection class	ArrayList

Name	Value
Show at runtime	true
Show name	true
Advanced	
Access type	public
Save in snapshot	true
Static	false
Description	
Description	Order: rc, rr, tc, tr, tt

Collection: cSDCadence

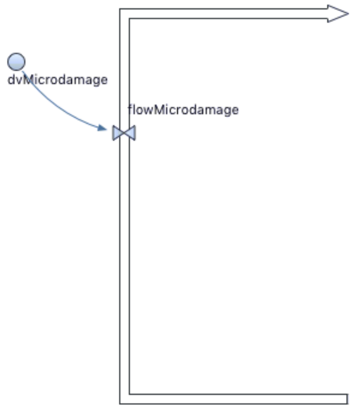
Description: Order: rc, rr, tc, tr, tt-1 , tt-2
(tt from separate cluster analysis)

From data analyses in R:
0.080008 0.077348 0.059396 0.095032 0.0712, 0.0605

Name	Value
General	
Initial contents	{0.08, 0.0773, 0.0594, 0.095, 0.0712, 0.0605}
Initial contents	{0.08, 0.0773, 0.0594, 0.095, 0.0712, 0.0605}
Element class	Double
Collection class	ArrayList
Show at runtime	true
Show name	true
Advanced	
Access type	public
Save in snapshot	true
Static	false
Description	
Description	Order: rc, rr, tc, tr, tt-1 , tt-2 (tt from separate cluster analysis) From data analyses in R: 0.080008 0.077348 0.059396 0.095032 0.0712, 0.0605

Collection: cCadenceLimits

Name	Value
General	
Initial contents	{3.00, 3.167}
Initial contents	{3.00, 3.167}
Element class	Double
Collection class	ArrayList
Show at runtime	true
Show name	true
Advanced	
Access type	public
Save in snapshot	true
Static	false

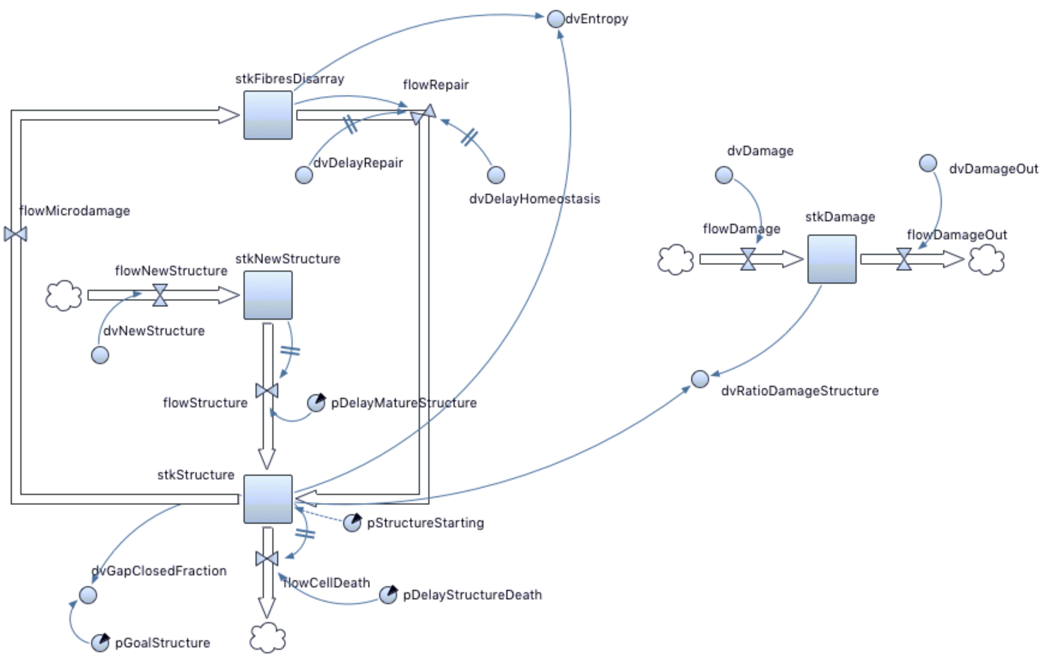


Dynamic Variable: dvMicrodamage

Name	Value
General	
Formula	fDriveMicrodamage()
Constant	false
External	false
Array	false
Show at runtime	true
Public	false
Show name	true
Advanced	
System dynamics units	false

Flow: flowMicrodamage

Name	Value
General	
Formula	fSwitchFlows() * dvMicrodamage
Constant	false
External	false
Array	false
Show at runtime	true
Public	false
Show name	true
Advanced	
System dynamics units	false



Stock: stkFibresDisarray

Name	Value
General	
Equation mode	Classic
Initial value	0
Array	false
Show at runtime	true
Public	false
Show name	true
Advanced	
System dynamics units	false

Flow: flowRepair

Name	Value
General	
Formula	fSwitchFlows() * (stkFibresDisarray / (dvDelayRepair + dvDelayHomeostasis))
Constant	false
External	false
Array	false
Show at runtime	true
Public	false
Show name	true
Advanced	
System dynamics units	false

Dynamic Variable: dvDelayRepair

Name	Value
General	
Formula	fDriveRecovery()
Constant	false
External	false
Array	false
Show at runtime	true
Public	false
Show name	true
Advanced	
System dynamics units	false

Dynamic Variable: dvEntropy

Name	Value
General	
Formula	abs(stkFibresDisarray / stkStructure)
Constant	false
External	false
Array	false
Show at runtime	true
Public	false
Show name	true
Advanced	
System dynamics units	false

Stock: stkStructure

Name	Value
General	
Equation mode	Classic
Initial value	pStructureStarting
Array	false
Show at runtime	true
Public	false
Show name	true
Advanced	
System dynamics units	false

Dynamic Variable: dvRatioDamageStructure

Name	Value
General	
Formula	abs(stkDamage / stkStructure)
Constant	false
External	false
Array	false
Show at runtime	true

Name	Value
Public	false
Show name	true
Advanced	
System dynamics units	false

Stock: stkDamage

Name	Value
General	
Equation mode	Classic
Initial value	0
Array	false
Show at runtime	true
Public	false
Show name	true
Advanced	
System dynamics units	false

Flow: flowCellDeath

Name	Value
General	
Formula	fSwitchFlows() * stkStructure / pDelayStructureDeath
Constant	false
External	false
Array	false
Show at runtime	true
Public	false
Show name	true
Advanced	
System dynamics units	false

Dynamic Variable: dvDelayHomeostasis

Name	Value
General	
Formula	fDriveHomeostasis()
Constant	false
External	false
Array	false
Show at runtime	true
Public	false
Show name	true
Advanced	
System dynamics units	false

Dynamic Variable: dvGapClosedFraction

Name	Value

Name	Value
General	
Formula	stkStructure / pGoalStructure
Constant	false
External	false
Array	false
Show at runtime	true
Public	false
Show name	true
Advanced	
System dynamics units	false

Stock: stkNewStructure

Name	Value
General	
Equation mode	Classic
Initial value	1
Array	false
Show at runtime	true
Public	false
Show name	true
Advanced	
System dynamics units	false

Flow: flowStructure

Name	Value
General	
Formula	fSwitchFlows() * stkNewStructure/ pDelayMatureStructure
Constant	false
External	false
Array	false
Show at runtime	true
Public	false
Show name	true
Advanced	
System dynamics units	false

Flow: flowNewStructure

Name	Value
General	
Formula	fSwitchFlows() * dvNewStructure
Constant	false
External	false
Array	false
Show at runtime	true
Public	false
Show name	true

Name	Value
Advanced	
System dynamics units	false

Dynamic Variable: dvNewStructure

Name	Value
General	
Formula	fDriveNewStructure()
Constant	false
External	false
Array	false
Show at runtime	true
Public	false
Show name	true
Advanced	
System dynamics units	false

Flow: flowRepair

Name	Value
General	
Formula	fSwitchFlows() * (stkFibresDisarray / (dvDelayRepair + dvDelayHomeostasis))
Constant	false
External	false
Array	false
Show at runtime	true
Public	false
Show name	true
Advanced	
System dynamics units	false

Flow: flowDamage

Name	Value
General	
Formula	fSwitchFlows() * dvDamage
Constant	false
External	false
Array	false
Show at runtime	true
Public	false
Show name	true
Advanced	
System dynamics units	false

Dynamic Variable: dvDamage

Name	Value
General	

Name	Value
Formula	fDriveDamage()
Constant	false
External	false
Array	false
Show at runtime	true
Public	false
Show name	true
Advanced	
System dynamics units	false

Flow: flowDamageOut

Name	Value
General	
Formula	fSwitchFlows() * dvDamageOut
Constant	false
External	false
Array	false
Show at runtime	true
Public	false
Show name	true
Advanced	
System dynamics units	false

Dynamic Variable: dvDamageOut

Name	Value
General	
Formula	fDriveDamageOut()
Constant	false
External	false
Array	false
Show at runtime	true
Public	false
Show name	true
Advanced	
System dynamics units	false

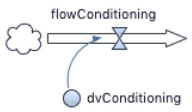
Flow: flowCellDeath

Name	Value
General	
Formula	fSwitchFlows() * stkStructure / pDelayStructureDeath
Constant	false
External	false
Array	false
Show at runtime	true
Public	false
Show name	true

Name	Value
Advanced	
System dynamics units	false

Flow: flowMicrodamage

Name	Value
General	
Formula	fSwitchFlows() * dvMicrodamage
Constant	false
External	false
Array	false
Show at runtime	true
Public	false
Show name	true
Advanced	
System dynamics units	false



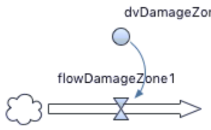
Dynamic Variable: dvConditioning

Name	Value
General	
Formula	fDriveConditioning()
Constant	false
External	false
Array	false
Show at runtime	true
Public	false
Show name	true
Advanced	
System dynamics units	false

Flow: flowConditioning

Name	Value
General	
Formula	fSwitchFlows() * dvConditioning
Constant	false
External	false
Array	false
Show at runtime	true
Public	false
Show name	true
Advanced	

Name	Value
System dynamics units	false

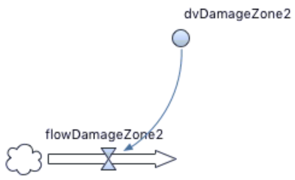


Dynamic Variable: dvDamageZone1

Name	Value
General	
Formula	fDriveDamageZone1()
Constant	false
External	false
Array	false
Show at runtime	true
Public	false
Show name	true
Advanced	
System dynamics units	false

Flow: flowDamageZone1

Name	Value
General	
Formula	fSwitchFlows() * dvDamageZone1
Constant	false
External	false
Array	false
Show at runtime	true
Public	false
Show name	true
Advanced	
System dynamics units	false



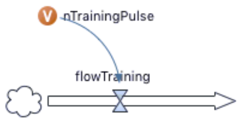
Dynamic Variable: dvDamageZone2

Name	Value
General	

Name	Value
Formula	fDriveDamageZone2()
Constant	false
External	false
Array	false
Show at runtime	true
Public	false
Show name	true
Advanced	
System dynamics units	false

Flow: flowDamageZone2

Name	Value
General	
Formula	fSwitchFlows() * dvDamageZone2
Constant	false
External	false
Array	false
Show at runtime	true
Public	false
Show name	true
Advanced	
System dynamics units	false



Flow: flowTraining

Description: The total impulse absorbed (integral of F(t) over time)

Name	Value
General	
Formula	fSwitchFlows() * fTrainingFlowSwitch() * nTrainingPulse
Constant	false
External	false
Array	false
Show at runtime	true
Public	false
Show name	true
Advanced	
System dynamics units	false
Description	
Description	The total impulse absorbed (integral of F(t) over time)

Stock: stkTrainingPulse

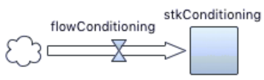
Description: Integration over time of the variable force (the flowTraining) gives the total impulse for the training session

Name	Value
General	
Equation mode	Classic
Initial value	0
Array	false
Color	lightSeaGreen
Show at runtime	true
Public	false
Show name	true
Advanced	
System dynamics units	false
Description	
Description	Integration over time of the variable force (the flowTraining) gives the total impulse for the training session

Flow: flowTraining

Description: The total impulse absorbed (integral of F(t) over time)

Name	Value
General	
Formula	fSwitchFlows() * fTrainingFlowSwitch() * nTrainingPulse
Constant	false
External	false
Array	false
Show at runtime	true
Public	false
Show name	true
Advanced	
System dynamics units	false
Description	
Description	The total impulse absorbed (integral of F(t) over time)



Stock: stkConditioning

Name	Value
General	
Equation mode	Classic

Name	Value
Initial value	0
Array	false
Show at runtime	true
Public	false
Show name	true
Advanced	
System dynamics units	false

Flow: flowConditioning

Name	Value
General	
Formula	fSwitchFlows() * dvConditioning
Constant	false
External	false
Array	false
Show at runtime	true
Public	false
Show name	true
Advanced	
System dynamics units	false

Stock: stkDamageZone1

Name	Value
General	
Equation mode	Classic
Initial value	0
Array	false
Show at runtime	true
Public	false
Show name	true
Advanced	
System dynamics units	false

Flow: flowDamageZone1

Name	Value
General	
Formula	fSwitchFlows() * dvDamageZone1
Constant	false
External	false
Array	false
Show at runtime	true

Name	Value
Public	false
Show name	true
Advanced	
System dynamics units	false

Stock: stkDamageZone2

Name	Value
General	
Equation mode	Classic
Initial value	0
Array	false
Show at runtime	true
Public	false
Show name	true
Advanced	
System dynamics units	false

Flow: flowDamageZone2

Name	Value
General	
Formula	fSwitchFlows() * dvDamageZone2
Constant	false
External	false
Array	false
Show at runtime	true
Public	false
Show name	true
Advanced	
System dynamics units	false

Dynamic Variable: dvTime

Name	Value
General	
Formula	time()
Constant	false
External	false
Array	false
Show at runtime	true
Public	false
Show name	true
Advanced	

Name	Value
System dynamics units	false

Dynamic Variable: dvGapStructure

Name	Value
General	
Formula	fDriveStructure()
Constant	false
External	false
Array	false
Show at runtime	true
Public	false
Show name	true
Advanced	
System dynamics units	false

Dynamic Variable: dvNewStructureMature

Name	Value
General	
Formula	fDriveNewStructureMature()
Constant	false
External	false
Array	false
Show at runtime	true
Public	false
Show name	true
Advanced	
System dynamics units	false

Dynamic Variable: dvDamageZone

Name	Value
General	
Formula	fDriveInjury()
Constant	false
External	false
Array	false
Show at runtime	true
Public	false
Show name	true
Advanced	
System dynamics units	false

Stock: stkTrainingLoad

Description: The cumulative training load

Name	Value
General	

Name	Value
Equation mode	Classic
Initial value	0
Array	false
Show at runtime	true
Public	false
Show name	true
Advanced	
System dynamics units	false
Description	
Description	The cumulative training load

Statechart Entry Point: stcTrainingStatus

Description: The purposes of the statechart:
Generate the intervals to sample from the pmf for the surface type

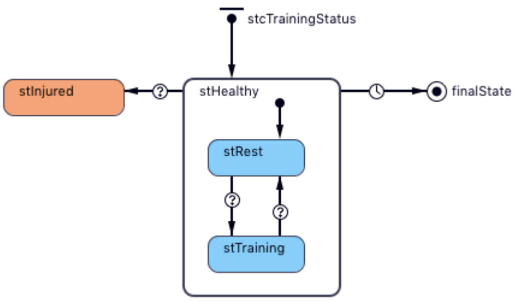
Reset the timer to count down to the next training session.
Capture the time when the current session ended.

Sample a new training parameters and biomechaincal metrics for each training session in the fTrainingParamters() function

Name	Value
General	
Logging	true

Action	//Generate the intervals to sample from the pmf for the surface type
	cIntervalsSurface.add(cPrbSurface.get(0));
	cIntervalsSurface.add(cPrbSurface.get(0) + cPrbSurface.get(1));
	cIntervalsSurface.add(cPrbSurface.get(0) + cPrbSurface.get(1) + cPrbSurface.get(2));
	cIntervalsSurface.add(cPrbSurface.get(0) + cPrbSurface.get(1) + cPrbSurface.get(2) + cPrbSurface.get(3));
	//cIntervalsSurface.add(cPrbSurface.get(0) + cPrbSurface.get(1) + cPrbSurface.get(2) + cPrbSurface.get(3) + cPrbSurface.get(4));
	
	//cIntervalsSurface.get(1)

Show at runtime	true
Show name	true
Description	
Description	The purposes of the statechart: Generate the intervals to sample from the pmf for the surface type Reset the timer to count down to the next training session. Capture the time when the current session ended. Sample a new training parameters and biomechaincal metrics for each training session in the fTrainingParamters() function



Transition: transition

Name	Value
General	
Action	fRecordOutput();
Condition	pDelayStructureDeath == 1;
Trigger type	Condition
Show name	false

Transition: transition1

Description: Exit at (2 x 12 months x 30 days per month)

Name	Value
General	
Action	fRecordOutput();
Timeout	(720 : DAY)
Trigger type	Timeout
Show name	false
Description	
Description	Exit at (2 x 12 months x 30 days per month)

Transition: transition2

Name	Value
General	
Action	//training starts
Condition	fTrainingFlowSwitch() != 0
Trigger type	Condition
Show name	false

Transition: transition3

Name	Value
General	
Action	//training ends
Condition	fTrainingFlowSwitch() == 0
Trigger type	Condition
Show name	false

State: stHealthy

Name	Value
General	

Entry action	//cIntervalsSurface.add(cPrbSurface.get(0));
	//cIntervalsSurface.add(cPrbSurface.get(0) + cPrbSurface.get(1));
	//cIntervalsSurface.add(cPrbSurface.get(0) + cPrbSurface.get(1) + cPrbSurface.get(2));
	//cIntervalsSurface.get(1)

Show name	true

State: stInjured

Description: Training stops

Name	Value
General	
Fill color	sandyBrown
Show name	true
Description	
Description	Training stops

State: stRest

Name	Value
General	

Entry action	nTrainingIntervalTime = pTrainingIntervalVariable * (1 - nDeltaTime) +
	pTrainingIntervalFixed * nTrainingIntervalFixed; //wait time to the next session
	
	//if(time() > 360 && time() < 720){
	//nMonth = 1
	//set training parameters for next session
	fTrainingParameters();
	fDamageControlCounter();
	fTrainingPulse();

Fill color	skyBlue
Show name	true

State: stTraining

Name	Value
General	

Exit action	//reset timer parameters
	
	nTrainingTimeEnded = time();
	
	nDeltaTime = nTrainingTimeEnded - nTrainingTimeStarted;
	
	//capture the total training pulse from the session and add to total training load
	stkTrainingLoad += stkTrainingPulse;
	datasetTrainingPulse.add(stkTrainingPulse);
	datasetTrainingload.add(stkTrainingLoad);
	datasetTrainingPulseSurface.add(pSurface);
	datasetTrainingTimeDuration.add(nDeltaTime);
	datasetTrainingIntervalTime.add(nTrainingIntervalTime); //the interval preceeding this training session
	datasetHeartRateZone.add(pHeartRateZone);
	
	//reset the training pulse to 0
	stkTrainingPulse = 0;

Entry action	nTrainingTimeStarted = time();
Fill color	skyBlue
Show name	true

Final State: finalState

Name	Value
General	

Name	Value
Show name	true

Initial State Pointer: initialState

Name	Value
General	
Show name	false

Time Plot: plotEntropyDamageStructure

Name	Value
General	
Lock	false
Public	true
Data update	
Analysis auto update	true
ANALYSIS_UPDATE_TIME_PROPERTIES	- Recurring Event Properties
Dataset Samples To Keep	10000
Scale	
Time window	1000
Time	model time units
Vertical scale	Auto
Appearance	
Labels horizontal position	DEFAULT
Labels vertical position	DEFAULT
Label format	Model time units
Labels Text Color	darkGray
Chart Area Grid Color	darkGray
Draw line	true
Fill area under line	true
Interpolation	Linear
Position and size	
x	130.0
Width	450.0
y	1080.0
Height	270.0
Legend	
Show legend	true
Legend size	30.0
Legend text color	black
Chart area	
Chart Area: X Offset	50.0
Chart Area: Width	370.0
Chart Area: Y Offset	30.0
Chart Area: Height	180.0
Chart Area: Background Color	white
Chart area border color	black
Advanced	

Name	Value
Time window moves	Continuously
Show name	false
Logging	true

Plot Items:

Title	Type	Dataset / Value	Point Style	Color	Line	Width	Interpolation
Entropy	value	dvEntropy	NONE	mediumSeaGreen	true	2.0	LINEAR
Damage:structure	value	dvRatioDamageStructure	NONE	slateBlue	true	2.0	LINEAR

Time Plot: plotGapClosed

Name	Value
General	
Lock	false
Public	true
Data update	
Analysis auto update	true
ANALYSIS_UPDATE_TIME_PROPERTIES	- Recurring Event Properties
Dataset Samples To Keep	7000
Scale	
Time window	720
Time	model time units
Vertical scale	Auto
Appearance	
Labels horizontal position	DEFAULT
Labels vertical position	DEFAULT
Label format	Model time units
Labels Text Color	darkGray
Chart Area Grid Color	darkGray
Draw line	true
Fill area under line	true
Interpolation	Linear
Position and size	
x	130.0
Width	980.0
y	1400.0
Height	360.0
Legend	
Show legend	true
Legend size	30.0
Legend text color	black
Chart area	
Chart Area: X Offset	50.0
Chart Area: Width	900.0
Chart Area: Y Offset	30.0
Chart Area: Height	270.0
Chart Area: Background Color	white
Chart area border color	black

Name	Value
Advanced	
Time window moves	Continuously
Show name	false
Logging	true

Plot Items:

Title	Type	Dataset / Value	Point Style	Color	Line	Width	Interpolation
Structure	value	dvGapClosedFraction	NONE	mediumTurquoise	true	2.0	LINEAR

Time Plot: plotTrainingLoad

Name	Value
General	
Lock	false
Public	true
Data update	
Analysis auto update	true
ANALYSIS_UPDATE_TIME_PROPERTIES	- Recurring Event Properties
Dataset Samples To Keep	1000
Scale	
Time window	300
Time	model time units
Vertical scale	Auto
Appearance	
Labels horizontal position	DEFAULT
Labels vertical position	DEFAULT
Label format	Model time units
Labels Text Color	darkGray
Chart Area Grid Color	darkGray
Draw line	true
Fill area under line	true
Interpolation	Linear
Position and size	
x	120.0
Width	840.0
y	860.0
Height	210.0
Legend	
Show legend	true
Legend size	30.0
Legend text color	black
Chart area	
Chart Area: X Offset	50.0
Chart Area: Width	760.0
Chart Area: Y Offset	30.0
Chart Area: Height	120.0
Chart Area: Background Color	white
Chart area border color	black

Name	Value
Advanced	
Time window moves	Continuously
Show name	false
Logging	true

Plot Items:

Title	Type	Dataset / Value	Point Style	Color	Line	Width	Interpolation
Training Load	value	stkTrainingLoad	NONE	darkMagenta	true	1.0	LINEAR

Link to agents: connections

Name	Value
General	
Show at runtime	true
Show name	true
Communication	
Message type	Object
Animation	
Draw line	false

Simulation Experiment: Simulation

Name	Value
General	
Maximum available memory	512
Agent type	Main
Model time	
Execution mode	Real time with scale
Real time scale	1.0
Stop option	Never
Initial time	0.0
Initial date	Tue Oct 20 00:00:00 GMT 2020
Randomness	
Random Number Generation Type	Fixed seed (reproducible simulation runs)
Seed value	1
Selection mode for simultaneous events	LIFO (in the reverse order of scheduling)
Window	
Title	RCASvisual2 : Simulation
Enable zoom and panning	true
Enable developer panel	true
Show developer panel on start	false
Advanced	
Load root from snapshot	false

RCASvisual2